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RESPIRATORY CARE

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SPECIAL ISSUE

SLEEP-DISORDERED BREATHING: PART II

CONFERENCE PROCEEDINGS

Non-Surgical Alternatives to Continuous Positive Airway Pressure

Surgical Therapy

Sleep-Disordered Breathing in Children

The Impact of Managed Care

Respiratory Care Practitioners and Sleep Medicine: Opportunities and Challenges

A Summary of the Conference

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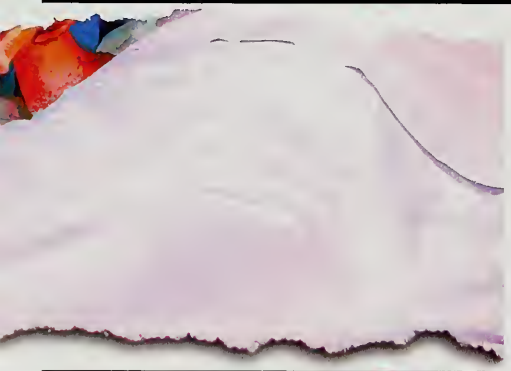

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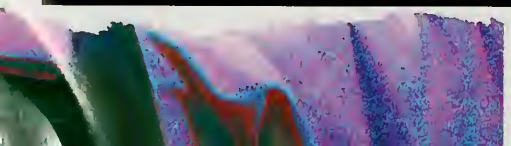

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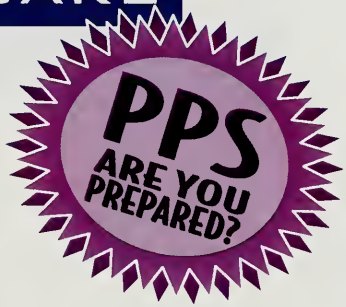

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RESPIRATORY CARE

A Monthly Science Journal Established in 1956

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SPECIAL ISSUE

SLEEP-DISORDERED BREATHING—PART II

The proceedings of a conference held in Cancún, Mexico, October 17-19, 1997

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Editorials, Commentaries, and Reviews To Note

The ABCs of Chest X-Ray Film Interpretation (Letter)—Crausman RS. Chest 1998;113(1): 256-257

Contamination of Doctors' and Nurses' Pens with Nosocomial Pathogens (Letter)—French G, Rayner D, Branson M, Walsh M. Lancet 1998;351(9097):213.

Descriptive Epidemiology of Asthma (Review)—Sears MR. Lancet 1997;350(Suppl 2):SII1-SII4. **Treatment of Acute Asthma** (Review)—Lipworth BJ. Lancet 1997;350(Suppl 2):SII18-SII23. **Limitations of Current Treatment** (Review)—Tattersfield AE. Lancet 1997;350(Suppl 2):SII24-SII27.

The Ethics of Bi-Level Positive Airway Pressure (Letter)—Crausman RS. Chest 1998;113(1):258.

Is It Time To Turn the Page on CLIA 1988? (Editorial)—Bachner P. JAMA 1998;279(6):473-475.

Jet-Lag (Letter)—Arendt J. Lancet 1998;351(9098):293-294 .

The Future of Asthma (Editorial)—Lancet 1997;350(9085):1113.

Update in Pulmonary Medicine (Review)—Niederman MS, Peters SP. Ann Intern Med 1998;128 (3):208-215.

Non-Invasive Diagnosis of *Pneumocystis carinii* Pneumonia (Letter)—Evans R, Ho-Yen DO. Lancet 1998;351(9097):218.

Cost-Effectiveness Analysis of Nocturnal Oximetry as a Method of Screening for Sleep Apnea-Hypopnea Syndrome—Epstein LJ, Dorlac GR. Chest 1998;113(1):97.

OBJECTIVE: Determine the utility of nocturnal oximetry as a screening tool for sleep apnea-hypopnea syndrome (SAHS) compared with polysomnography (PSG). DESIGN: Cost-effectiveness analysis based on retrospective review of overnight sleep studies. SETTING: United States Air Force tertiary teaching hospital. PATIENTS: One hundred consecutive patients evaluated for SAHS by overnight sleep study. INTERVENTION: Participants underwent PSG and oximetry on the same night. Patients with obstructive sleep apnea had a continuous positive airway pressure trial. MEASUREMENTS: Oximetry was abnormal when ≥ 10 events/hour occurred. Two criteria were evaluated: a "deep" pattern of > 4% change in oxyhemoglobin saturation to l90% and a "fluctuating" pattern of repetitive short-duration fluctuations in saturation. The diagnostic accuracy of both methods was compared with PSG. Cost-effectiveness of screening oximetry was compared with PSG alone and use of split-night studies. RESULTS: The fluctuating pattern had a greater sensitivity and negative predictive value, while the deep pattern had a greater specificity and positive predictive value. Oximetry screening using the fluctuating pattern was not as sensitive as PSG for detecting patients with mild disease; 17 of 28 patients (61%) with normal oximetry results had treatable conditions detected by PSG. Cost analysis showed that screening oximetry would save $4,290/100 patients but with considerable loss of diagnostic accuracy. CONCLUSION: Screening oximetry is not cost-effective because of poor diagnostic accuracy despite increased sensitivity using the fluctuating pattern. Greater savings, without loss of diagnostic accuracy, may be achieved through increased utilization of split-night PSGs.

Cigarette Smoking and Progression of Atherosclerosis: The Atherosclerosis Risk in Communities (ARIC) Study—Howard G, Wagenknecht LE, Burke GL, Diez-Roux A, Evans GW, McGovern P, Nieto FJ, Tell GS. JAMA 1998; 279(2):119.

CONTEXT: Cigarette smoking is a powerful risk factor for incident heart disease and stroke, but the relationship of active and passive smoking with the progression of atherosclerosis has not been described. OBJECTIVE: To examine the impact of active smoking and exposure to environmental tobacco smoke (ETS) on the progression of atherosclerosis. DESIGN: A longitudinal assessment of the relationship between smoking exposure evaluated at the initial visit and the 3-year change in atherosclerosis. SETTING: A population-based cohort of middle-aged adults from 4 communities in the United States. PARTICIPANTS: A total of 10,914 participants from the Atherosclerosis Risk in Communities (ARIC) study enrolled between 1987 and 1989. MAIN OUTCOME MEASURE: Change in atherosclerosis from baseline to the 3-year follow-up as indexed by intimal-medial thickness of the carotid artery assessed by ultrasound and adjusted for demographic characteristics, cardiovascular risk factors, and lifestyle variables. RESULTS: Exposure to cigarette smoke was associated with progression of atherosclerosis. Relative to never smokers and after adjustment for demographic characteristics, cardiovascular risk factors, and lifestyle variables, current cigarette smoking was associated with a 50% increase in the progression of atherosclerosis (mean progression rate over 3 years, 43.0 μm for current and 28.7 μm for never smokers, regardless of ETS exposure), and past smoking was associated with a 25% increase (mean progression rate over 3 years, 35.8 μm for past smokers and 28.7 μm for never smokers). Relative to those not exposed to ETS, exposure to ETS was associated with a 20% increase (35.2 μm for those exposed to ETS vs 29.3 μm for those not

exposed). The impact of smoking on atherosclerosis progression was greater for subjects with diabetes and hypertension. Although more pack-years of exposure was independently associated with faster progression ($p < 0.001$), after controlling for the number of pack-years, the progression rates of current and past smokers did not differ ($p = 0.11$). CONCLUSIONS: Both active smoking and ETS exposure are associated with the progression of an index of atherosclerosis. Smoking is of particular concern for patients with diabetes and hypertension. The fact that pack-years of smoking but not current versus past smoking was associated with progression of atherosclerosis suggests that some adverse effects of smoking may be cumulative and irreversible.

Trends in Pediatric Asthma Hospitalization Rates: Regional and Socioeconomic Differences—Goodman DC, Stukel TA, Chang CH. Pediatrics 1998;101(2):208.

OBJECTIVE: Asthma hospitalization rates continue to increase nationally for children despite efforts by the National Institutes of Health and specialty organizations to improve outcomes through the dissemination of practice guidelines. To understand the generalizability of national trends to regional populations, we studied childhood hospitalizations over a 10-year period in 4 northeastern states. DESIGN: Longitudinal analysis of hospitalization rates by patient residence and patient characteristics using state hospital discharge data sets. POPULATION: Age < 18 years residing in Maine, New Hampshire, Vermont, or New York state during the period 1985 to 1994. RESULTS: In multivariate analyses (controlling for age, sex, race/ethnicity, median household income, metropolitan status), we found that New York asthma hospitalization rates increased 3.8%/annum (95% confidence interval [CI]: 3.3, 4.2), whereas in New Hampshire, rates decreased 5.8% (95% CI: 7.6, 4.1). Maine and Vermont rates did not change significantly during the study period. Increased asthma hospitalization rates were noted in black and Hispanic populations, in children residing in zip codes with lower median household incomes, and in those living in metropolitan areas. Hospitalization rates for nonasthma causes fell substantially. As a result, the proportion of hospital days attributed to childhood asthma increased in all population groups. CONCLUSIONS: Asthma discharge rates measured by the state of residence or socioeconomic characteristic do not necessarily parallel national trends. None of the current hypotheses offered to explain national trends in asthma hospitalization rates (changes in disease severity, diagnostic substitution, or differences in the supply and character of medical care) can be the sole explanation of these regional trends. Efforts intended to improve asthma outcomes may benefit a greater number of children by redirecting resources toward specific populations identified through state hospital discharge datasets.

Partial Liquid Ventilation with Perfluorocarbon in the Treatment of Rats with Lethal Pneumococcal Pneumonia—Dickson EW, Heard SO, Chu B, Fraire A, Brueggemann AB, Doern GV. Anesthesiology 1998;88(1):218.

BACKGROUND: Partial liquid ventilation with perfluorocarbon is a new therapeutic strategy to treat various lung disorders. The current study was undertaken to determine the efficacy of partial liquid ventilation with a perfluorocarbon (FC-77) in the treatment of pneumococcal pneumonia in rats. METHODS: Male Wistar rats (weight, 275-300 g; n, 75) were infected via direct intratracheal inoculation with ca 10^9 colony-forming units of viable *Streptococcus pneumoniae*, serotype 3, and 24 hours after infection were placed into 1 of 5 groups, each containing 15 rats. The groups were (1) no treatment, (2) 1 intramuscular injection of penicillin G benzathine (200,000 U), (3) partial liquid ventilation with FC-77, (4) partial liquid ventilation with FC-77 and a single intramuscular dose of penicillin G benzathine (200,000 U), and (5) gas ventilation. Animals were observed every 24 hours for survival. RESULTS: All untreated or gas-ventilated animals or animals that received only partial liquid ventilation were dead by 7 days. Those receiving only partial liquid ventilation survived longer than untreated controls, but ultimately all succumbed by Day 7. Survival was 40% for penicillin-treated rats compared with controls ($p < 0.05$) and 80% for animals treated with both partial liquid ventilation and penicillin versus antibiotic alone ($p < 0.05$). CONCLUSIONS: These observations suggest that partial liquid ventilation with perfluorocarbon in combination with antibiotic administration may be an effective therapeutic modality in pneumococcal pneumonia.

Effect of Inverse I:E Ratio Ventilation on Pulmonary Gas Exchange in Acute Respiratory Distress Syndrome—Zavala E, Ferrer M, Polese G, Masclans JR, Planas M, Milic-Emili J, Rodriguez-Roisin R, Roca J, Rossi A. Anesthesiology 1998;88(1):35.

BACKGROUND: It is not known whether inverse I:E ratio ventilation (IRV) offers any real benefit over conventional mechanical ventilation with positive end-expiratory pressure (CMV-PEEP) at similar levels of end-expiratory pressure. METHODS: The effects of volume-controlled and pressure-controlled IRV (VC-IRV and PC-IRV, respectively) on $\dot{V}_A/\dot{Q}$ inequality were compared with those of CMV-PEEP at a similar level of end-expiratory pressure and with CMV without PEEP (CMV) in 8 patients in the early stages of acute respiratory distress syndrome (ARDS). Respiratory blood gases, inert gases, lung mechanics, and hemodynamics were measured 30 minutes after the onset of each ventilatory mode. RESULTS: Recruitment of nonventilated, poorly ventilated (or both) but well-perfused alveoli increased the partial pressure of oxygen (P_{aO_2}) during CMV-PEEP (+13 mm Hg) and IRV-VC (+10 mm Hg; $p < 0.05$) compared with CMV. In contrast, PC-IRV did not affect P_{aO_2} but caused a decrease in P_{aCO_2} (−7 mm Hg; $p < 0.05$). The latter was due to a concomitant decrease in dead space ($p < 0.01$) and shift to the right of $\dot{V}_A/\dot{Q}$ distributions. During PC-IRV, the increase in the mean of blood flow distribution (mean $\dot{Q}$; $p < 0.01$) without a change in the dispersion (log SD $\dot{Q}$) did not result in an increase in P_{aO_2}, probably because it reflected redistribution of blood flow within well-ventilated areas. CONCLUSIONS: Short-term PC-IRV improved carbon dioxide clearance, but the lung became less efficient as an oxygen exchanger. Furthermore, based on mean airway and plateau pressures, the risk of barotrauma was not reduced with this type of ventilation.

Transudative Malignant Pleural Effusions: Prevalence and Mechanisms—Ashchi M, Golish J, Eng P, O'Donovan P. South Med J 1998; 91(1):23-26

BACKGROUND: Malignant pleural effusions are classically exudative, though occasionally they can be transudative. We attempted to determine the prevalence of transudative malignant pleural effusions and to describe their clinical and radiographic characteristics. METHODS: We studied the medical records of 215 patients with malignant pleural effusion, which contained complete data on pleural fluid analysis for 171 of them. RESULTS: Eight cases of transudative malignant effusion were found. Seven of these 8 patients had a concomitant disorder known to cause transudative pleural effusion. One patient had fluid overload due to renal failure, and another had been admitted earlier with congestive heart failure. Two patients had definite evidence of deep venous thrombosis, allowing us to postulate pulmonary embolism as a factor contributing to transudation. Three patients had computed tomography evidence of superior vena cava obstruction. Only 1 patient had no known associations with causes of transudative effusion. CONCLUSION: Malignant pleural effusions were transudative in 5% of our patients. In all except 1 patient, some evidence suggested the concomitant presence of a transudative state.

Risky Behavior in Teens with Cystic Fibrosis or Sickle Cell Disease: A Multicenter Study—Britto MT, Garrett JM, Dugliss MA, Daeschner CW Jr, Johnson CA, Leigh MW, Majure JM, Schultz WH, Konrad TR. Pediatrics 1998;101 (2):250.

OBJECTIVE: To determine the prevalence and age of onset of common risky behaviors such as smoking and sexual activity in teens with cystic fibrosis and those with sickle cell disease and to compare their behaviors with those of adolescents in the general population. DESIGN: Survey. SETTING: All 5 major pediatric tertiary care centers

in North Carolina (study participants with sickle cell disease or cystic fibrosis) and North Carolina public schools (comparison population). PARTICIPANTS: Three hundred twenty-one adolescents with cystic fibrosis or sickle cell disease aged 12 to 19 years (mean age, 15.6 years; 49% female). Demographically matched comparison teens for each group were selected from 2760 in-school adolescents (mean age, 16.0 years; 51% female). MAIN OUTCOMES MEASURES: Prevalence of tobacco and marijuana use, alcohol use, sexual intercourse, sexually transmitted diseases, seat belt use, weapon carrying, and age of onset of these behaviors. RESULTS: Chronically ill teens reported significantly less lifetime and current use of tobacco, marijuana, and alcohol; less sexual intercourse; less weapon carrying, less drunk driving, and more seat belt use than their peers. Nonetheless, 21% of the teens with cystic fibrosis and 30% of those with sickle cell disease had smoked; sexual intercourse was reported by 28% and 51%, respectively. Age of onset of these behaviors was frequently older for the chronically ill teens. CONCLUSION: Teens with cystic fibrosis or sickle cell disease took more potentially damaging health risks than might be expected, although the prevalence was lower than reported by their peers. Future longitudinal studies should examine the relationships between chronic illness, physical and psychosocial maturation, and risky behavior. Screening for psychosocial issues, including risky behaviors, should be incorporated into the routine health care of chronically ill teens.

Evidence of a Critical Period of Airway Instability during Central Apneas in Preterm Infants—Lemke RP, Idiong N, Al-Saedi S, Kwiatkowski K, Cates DB, Rigatto H. Am J Respir Crit Care Med 1998;157(2):470.

The timing and magnitude of airway narrowing in central apneas is unknown. We have developed a method of apnea classification that relies on the transmission of cardiac airflow oscillation to indicate airway patency. Using a theoretical model, we showed that the amplitude of the cardiac airflow oscillation is proportional to airway diameter for small lumens. While in the majority of central apneas the amplitude of the cardiac airflow oscillation remains nearly constant, in a subset of events the waveform decreases with time, suggesting airway narrowing. We hypothesized that this is not a random occurrence but reflects a critical period of airway instability during central apnea. To test this hypothesis we studied 41 preterm infants. Of 4,456 central apneas, 585 had a decrease in the amplitude of the cardiac oscillation. The amplitude of the cardiac airflow oscillation during an apnea was recorded to provide a dynamic measure of changes in airway diameter with time. To allow for comparisons between patients the amplitude of each cardiac airflow oscillation was expressed as a proportion of the maximum amplitude observed in each infant. We then compared the amplitude at multiple successive 0.5 s intervals with the amplitude of the cardiac airflow oscillation observed at the apnea outset using ANOVA. We found a significant decrease in cardiac airflow oscillation after only 1 s irrespective of the apnea duration (3 to 16 s). We conclude that airway narrowing during central apnea is not a random occurrence but appears shortly after the onset of the apnea. We speculate that the phenomenon is secondary to passive airway relaxation.

Pulmonary Infiltrates, Eosinophilia, and Cardiomyopathy following Corticosteroid Withdrawal in Patients with Asthma Receiving Zafirlukast—Wechsler ME, Garpestad E, Flier SR, Kocher O, Weiland DA, Polito AJ, Klinek MM, Bigby TD, Wong GA, Helmers RA, Drazen JM. JAMA 1998;279(6):455.

CONTEXT: Zafirlukast is a potent leukotriene antagonist that recently was approved for the treatment of asthma. As use of this drug increases, adverse events that occur at low frequency or in populations not studied in premarketing clinical trials may become evident. OBJECTIVE: To describe a clinical syndrome associated with zafirlukast therapy. DESIGN: Case series. PATIENTS: Eight adults (7 women and 1 man) with steroid-dependent asthma who received zafirlukast. MAIN OUTCOME MEASURES: Development of a clinical syndrome characterized by pulmonary infiltrates, cardiomyopathy, and eosinophilia following the withdrawal of corticosteroid treatment. RESULTS: The clinical syndrome developed while patients were receiving zafirlukast from 3 days to 4 months and from 3 days to 3 months after corticosteroid withdrawal. All 8 patients developed leukocytosis (range, 14.5-27.6 M 109/L) with eosinophilia (range, 0.19-0.71). Six patients had fever (temperature > 38.5° C), 7 had muscle pain, 6 had sinusitis, and 6 had biopsy evidence of eosinophilic tissue infiltration. The clinical syndrome improved with discontinuation of zafirlukast treatment and reinitiation of corticosteroid treatment or addition of cyclophosphamide treatment. COMMENT: Development of pulmonary infiltrates, cardiomyopathy, and eosinophilia may have occurred independent of zafirlukast use or may have resulted from an allergic response to this medication. We suspect that these patients may have had a primary eosinophilic infiltrative disorder that had been clinically recognized as asthma, was quelled by steroid treatment, and was unmasked following corticosteroid withdrawal facilitated by zafirlukast.

Health Values of Hospitalized Patients 80 Years or Older. HELP Investigators. Hospitalized Elderly Longitudinal Project—Tsevat J, Dawson NV, Wu AW, Lynn J, Soukup JR, Cook EF, Vidaillet H, Phillips RS. JAMA 1998;279(5):371.

CONTEXT: Health values (utilities or preferences for health states) are often incorporated into clinical decisions and health care policy when issues of quality versus length of life arise, but little is known about health values of the very old. OBJECTIVE: To assess health values of older hospitalized patients, compare their values with those of their surrogate decision makers, investigate possible determinants of health values, and determine whether health values change over time. DESIGN: A prospective, longitudinal, multicenter cohort study. SETTING: Four academic medical centers. PARTICIPANTS: Four hundred fourteen hospitalized patients aged 80 years or older and their surrogate decision makers who were interviewed and understood the task. MAIN OUTCOME MEASURES: Time-trade-off utilities, reflecting preferences for current health relative to a shorter but healthy life. RESULTS: On average, patients equated living 1 year in their current state of health with living 9.7 months in excellent health (mean [standard deviation (SD)] utility, 0.81 [0.28]). Although only 126 patients (30.7%) rated their current quality of life as excellent or very good, 284 (68.6%) were willing to give up at most 1 month of 12 in exchange for excellent health (utility ≥ 0.92). At the other extreme, 25 (6.0%) were willing to live 2 weeks or less in excellent health rather than 1 year in their current state of health (utility ≤ 0.04). Patients were willing to trade significantly less time for a healthy life than their surrogates assumed they would (mean difference, 0.05; $p = 0.007$); 61 surrogates (20.3%) underestimated the patient's time-trade-off score by 0.25 (3 months of 12) or more. Patients willing to trade less time for better health were more likely to want resuscitation and other measures to extend life. Time-trade-off score correlated only modestly with quality-of-life rating ($r = 0.28$) and inversely with depression score ($r = -0.27$), but there were few other clinical or demographic predictors of health values. When patients who survived were asked the time-trade-off question again at 1 year, they were willing to trade less time for better health than at baseline (mean difference, 0.04; $p = 0.04$). CONCLUSION: Very old hospitalized patients who could be interviewed were able, in most cases, to have their health values assessed using the time-trade-off technique. Most patients were unwilling to trade much time for excellent health, but preferences varied greatly. Because proxies and multivariable analyses cannot gauge health values of elderly hospitalized patients accurately, health values of the very old should be elicited directly from the patient.

Safety of Hospitalized Ventilator-Dependent Children Outside of the Intensive Care Unit—Ambrosio JU, Woo MS, Jansen MT, Keens TG. Pediatrics 1998;101(2):257.

OBJECTIVE: Hospitalization of clinically stable ventilator-dependent children in an intensive care unit (ICU) remains the standard in most pediatric centers. The aim of this study was to determine whether chronically ventilator-depen-

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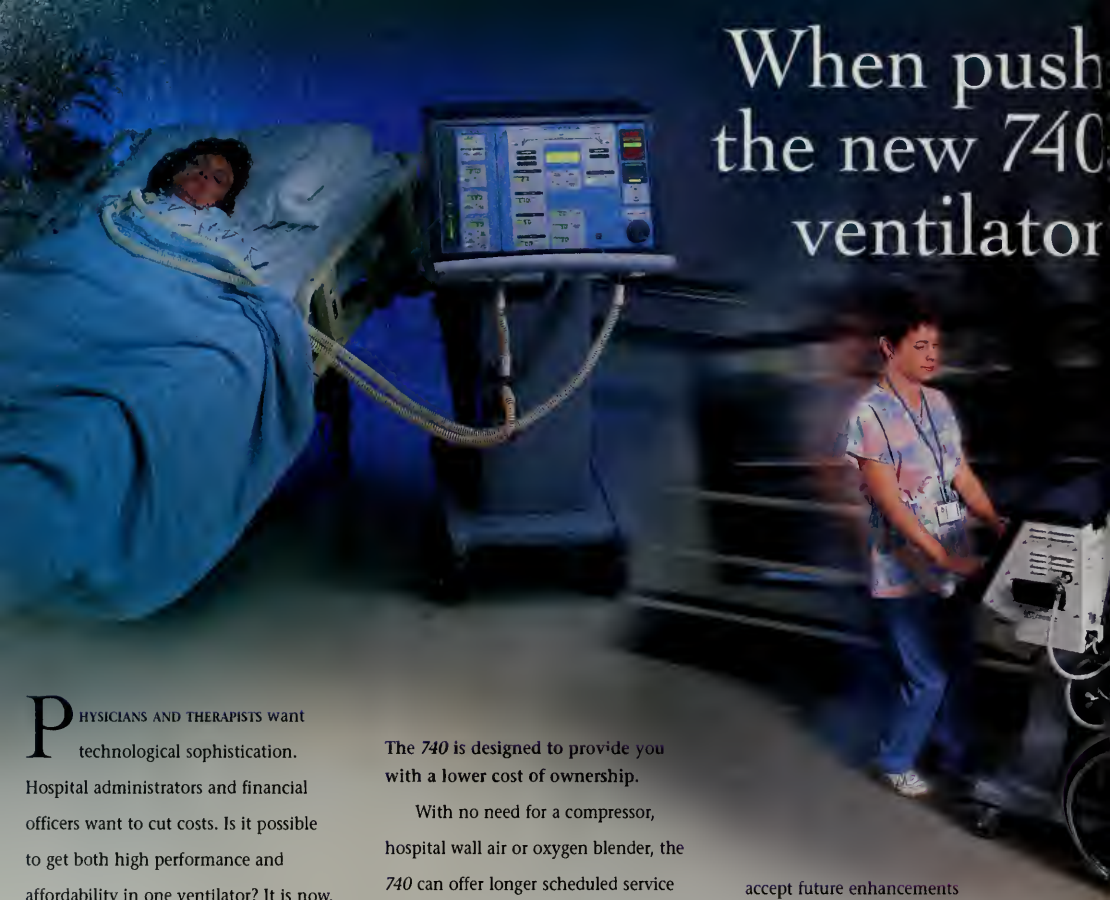

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T O R S

dent children could be hospitalized safely in a non-ICU setting. METHODS: All ventilator-dependent children who were hospitalized on the pediatric wards at Childrens Hospital Los Angeles from December 1992 through June 1996 were reviewed retrospectively (n = 63) and compared with the general pediatric ward population hospitalized during the same period. Data collected included the number of unexpected ICU transfers from the pediatric ward and the number of deaths that occurred on the ward. RESULTS: The ventilator-dependent children on the pediatric wards had 11 emergency readmissions to the ICU for unexpected deterioration. This represented an unexpected ICU transfer rate of 2.7/1,000 patient-days on the wards. The general pediatric ward population had an unexpected ICU transfer rate of 3.3/1,000 patient-days, which was not significantly different from that of ventilator-dependent children on the wards. There were 3 ward deaths among the ventilator-dependent children, but all of these patients had advance directive status (do not resuscitate). This represented a rate of 7 deaths/10,000 patient-days on the wards, which was not significantly different from those of nonventilator-dependent ward patients (8 deaths/10,000 patient-days). CONCLUSIONS: We conclude that ventilator-dependent children hospitalized outside of the ICU do not have an increased incidence of deaths and unexpected ICU admissions compared with nonventilator-dependent inpatients. We speculate that hospital care of stable ventilator-dependent children can be provided safely outside of an ICU and at lower cost.

Pulmonary Vasodilation by Nitric Oxide Gas and Prodrug Aerosols in Acute Pulmonary Hypertension—Adrie C, Ichinose F, Holzmann A, Keefer L, Hurford WE, Zapol WM. J Appl Physiol 1998;84(2):435.

Sodium 1-(N,N-diethylamino)diazen-1-ium-1,2-diolate {DEA/NO; Et2N[N(O)NO]Na} is a compound that spontaneously generates nitric oxide (NO). Because of its short half-life (2.1 min), we hypothesized that inhaling DEA/NO aerosol would selectively dilate the pulmonary circulation without decreasing systemic arterial pressure. We compared the pulmonary selectivity of this new NO donor with 2 other reference drugs: inhaled NO and inhaled sodium nitroprusside (SNP). In 7 awake sheep with pulmonary hypertension induced by the infusion of U-46619, we compared the hemodynamic effects of DEA/NO with those of incremental doses of inhaled NO gas. In 7 additional awake sheep, we examined the hemodynamic effects of incremental doses of inhaled nitroprusside (ie, SNP). Inhaled NO gas selectively dilated the pulmonary vasculature. Inhaled DEA/ NO produced nonselective vasodilation; both systemic vascular resistance (SVR) and pulmonary vascular resistance (PVR) were reduced. Inhaled SNP selectively dilated the pulmonary circulation at low concentrations ($\leq 10^{-2}$ M), inducing a decrease of PVR of up to 42% without any significant decrease of SVR (−5%), but nonselectively dilated the systemic circulation at larger doses ($> 10^{-2}$ M). In conclusion, despite its short half-life, DEA/NO is not a selective pulmonary vasodilator compared with inhaled NO. Inhaled SNP appears to be selective to the pulmonary circulation at low doses but not at higher levels.

Effects of Flow Triggering on Breathing Effort during Partial Ventilatory Support—Aslanian P, El Atrous S, Isabey D, Valente E, Corsi D, Harf A, et al. Am J Respir Crit Care Med 1998;157 (1):135.

The effects of flow triggering (FT) as compared with pressure triggering (PT) on breathing effort have been the focus of several studies, and discrepant results have been reported. In the initial part of our study, a lung model was used to quantify triggering effort (airway pressure-time product, PTP_{aw}) for a range of sensitivity settings in 9 new-generation ventilators. A ventilator providing both FT and PT was then used to compare these systems during pressure-support (PSV) and volume-targeted assist-control ventilation (ACV) in 8 ventilator-dependent patients, using sensitivity settings (2 L/min for FT and −2 cm H_2O for PT) that had proven significantly different in the initial bench study. Indexes of effort included the esophageal and transdiaphragmatic pressure-time products and inspiratory work of breathing per minute (PTP_{es}/min, PTP_{di}/min, and W_i/min, respectively). The experimental study revealed significant differences between ventilators in PTP_{aw} at commonly used settings. In 2 of 3 ventilators featuring both systems, PTP_{aw} was significantly lower with FT than PT ($p < 0.001$). In the clinical study, FT as compared with PT, was associated with reductions in all indexes of breathing effort during PSV: 16 ± 6% ($p < 0.001$), 13 ± 10% ($p < 0.01$), and 14 ± 12% ($p < 0.05$) for PTP_{di}/min, PTP_{es}/min, and W_i/min, respectively. By contrast, no differences were found when FT was used during ACV. Although FT reduced triggering effort in both modes ($p < 0.001$), the effects observed during the post-trigger phase differed, and explained the discrepant results between the 2 modes. We conclude that FT more effectively reduces breathing effort when used in conjunction with a pressure-targeted mode than with a volume-targeted mode, especially when flow delivery is close to or below demand.

Impact of Clinical Pathways and Practice Goidelines on the Management of Acute Exacerbations of Bronchial Asthma—Bailey R, Weingarten S, Lewis M, Mohsenifar Z. Chest 1998;113(1):28.

OBJECTIVES: In 1990, it was estimated that approximately 1% of all U.S. health care costs (approximately $6.2 billion) were spent on asthma-related health expenses. Of this, hospitalization charges alone exceeded $2.6 billion. Practice guidelines and clinical pathways are being developed to standardize the management of acute asthma with the aim of improving care and safely reducing health care costs. In this report, we evaluate the impact of an asthma pathway developed and instituted at a large community-based teaching hospital. This pathway was evidence based and was developed by a multidisciplinary group. METHODS: The study was conducted during a 6-month period in 1995, while a similar period in 1994 was used as a historical control period. Data collected included patient demographics, hospital admission and discharge peak expiratory flow rates, pulse oximetry measurements, length of stay, conversion from hand-held nebulizer to metered dose inhaler, use of corticosteroids within 24 hours of hospitalization, and conversion of I.V. steroids to oral steroids. RESULTS: A total of 42 patients were enrolled during the study period. Of these, 19 were placed on the pathway, while 23 were not treated according to the pathway. There were 38 patients in the 1994 historical control period. For 1995, there was no significant difference between the pathway and nonpathway groups with regard to the length of stay (4.4 ± 3.3 vs 3.2 ± 2.3 days; $p > 0.05$), hospital discharge peak expiratory flow rates (324 vs 286 L/min; $p > 0.05$), or use of steroids (100% vs 91%; $p > 0.05$). However, a significant increase in conversion from hand-held nebulizer to metered dose inhaler was noted in the pathway group (68% vs 34%; $p < 0.05$). The data from 1994 compared to 1995 pathway were similar in that there was no difference in the length of stay (3.4 ± 2.1 vs 4.4 ± 3.3 days; $p > 0.05$) and/ or use of steroids (92% vs 100%; $p > 0.05$), while a significant increase in hand-held nebulizer to metered dose inhaler conversion was observed for the 1995 pathway group (68% vs 26%; $p = 0.002$). CONCLUSIONS: We conclude that although the asthma pathway did not significantly reduce length of stay, it was associated with a significant increase in hand-held nebulizer to metered dose inhaler conversion, resulting in a substantial cost savings of $288,000/year.

Effect of a Protective-Ventilation Strategy on Mortality in the Acute Respiratory Distress Syndrome—Amato MB, Barbas CS, Medeiros DM, Magaldi RB, Schettino GP, Lorenzi-Filho G, et al. N Engl J Med 1998;338(6):347.

BACKGROUND: In patients with the acute respiratory distress syndrome (ARDS), massive alveolar collapse and cyclic lung reopening and overdistention during mechanical ventilation may perpetuate alveolar injury. We determined whether a ventilatory strategy designed to minimize such lung injuries could reduce not only pulmonary complications but also mortality at 28 days in patients with ARDS. METHODS: We randomly assigned 53 patients with early ARDS (including 28 described previously), all of whom were receiv-

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ing identical hemodynamic and general support, to conventional or protective mechanical ventilation. Conventional ventilation was based on the strategy of maintaining the lowest positive end-expiratory pressure (PEEP) for acceptable oxygenation, with a tidal volume of 12 mL/kg of body weight and normal arterial carbon dioxide levels (35 to 38 mm Hg). Protective ventilation involved end-expiratory pressures above the lower inflection point on the static pressure-volume curve, a tidal volume of < 6 mL/kg, driving pressures of < 20 cm H_2O above the PEEP value, permissive hypercapnia, and preferential use of pressure-limited ventilatory modes. RESULTS: After 28 days, 11 of 29 patients (38%) in the protective-ventilation group had died, as compared with 17 of 24 (71%) in the conventional-ventilation group ($p < 0.001$). The rates of weaning from mechanical ventilation were 66% in the protective-ventilation group and 29% in the conventional-ventilation group ($p = 0.005$): the rates of clinical barotrauma were 7% and 42%, respectively ($p = 0.02$), despite the use of higher PEEP and mean airway pressures in the protective-ventilation group. The difference in survival to hospital discharge was not significant; 13 of 29 patients (45%) in the protective-ventilation group died in the hospital, as compared with 17 of 24 in the conventional-ventilation group (71%, $p = 0.37$). CONCLUSIONS: As compared with conventional ventilation, the protective strategy was associated with improved survival at 28 days, a higher rate of weaning from mechanical ventilation, and a lower rate of barotrauma in patients with ARDS. Protective ventilation was not associated with a higher rate of survival to hospital discharge.

Antenatal Thyrotropin-Releasing Hormone To Prevent Lung Disease in Preterm Infants. North American Thyrotropin-Releasing Hormone Study Group—Ballard RA, Ballard PL, Cnaan A, Pinto-Martin J, Davis DJ, Padbury JF, et al. N Engl J Med 1998;338(8):493.

BACKGROUND: Pulmonary disease is common in preterm infants, despite antenatal glucocorticoid therapy. The addition of antenatal thyrotropin-releasing hormone therapy has been reported to decrease pulmonary morbidity in these infants. METHODS: We enrolled 996 women at 13 North American centers who were in preterm labor at < 30 weeks' gestation in a double-blind, placebo-controlled, randomized trial of antenatal thyrotropin-releasing hormone, given intravenously in 4 doses of 400 μg each at 8-hour intervals. The primary outcome was chronic lung disease or death of the infant on or before the 28th day after delivery, and secondary outcomes were respiratory distress syndrome and chronic lung disease or death at 36 weeks' postmenstrual age. Complete data were available for 981 women and their 1,134 live-born infants. The 769 infants born at ≤ 32 weeks' gestation were defined as the group at risk. RESULTS: There were no significant differences between the at-risk treatment and placebo groups in mean (± SD) birthweight (1,109 ± 354 vs 1,097 ± 355 g), gestational age (27.9 ± 2.1 vs 27.9 ± 2.1 weeks), sex, or race. The frequencies of respiratory distress syndrome (66% vs 65%), death at 28 days (11% vs 11%), chronic lung disease or death at 28 days (45% vs 42%) and at 36 weeks (32% vs 34%), and other neonatal complications as well as the severity of lung disease were not significantly different in the at-risk treatment and placebo groups. Similarly, there were no differences in outcome between the treatment and placebo groups for the infants born at > 32 weeks' gestation. CONCLUSIONS: In preterm infants at risk for lung disease, antenatal administration of thyrotropin-releasing hormone and glucocorticoid is no more beneficial than glucocorticoid alone.

Temporal Hemodynamic Effects of Permissive Hypercapnia Associated with Ideal PEEP in ARDS—Carvalho CR, Barbas CS, Medeiros DM, Magaldi RB, Lorenzi Filho G, Kairalla RA, et al. Am J Respir Crit Care Med 1997;156(5):1458.

The associated use of permissive hypercapnia (PHY) and high PEEP levels ($PEEP_{IDEAL}$) has

been recently indicated as part of a lung-protective-approach (LPA) in acute respiratory distress syndrome (ARDS). However, the net hemodynamic effect produced by this association is not known. We analyzed the temporal hemodynamic effects of this combined strategy in 48 patients (mean age 34 ± 13 yr) with ARDS, focusing on its immediate (after 1 h), early (first 36 h), and late (2nd-7th d) consequences. Twenty-five patients were submitted to LPA—with the combined use of permissive hypercapnia (PHY), $V_T < 6$ mL/kg, distending pressures above PEEP < 20 cm H_2O, and PEEP 2 cm H_2O above the lower inflection point on the static inspiratory P-V curve (P_{FLEX}) and 23 control patients were submitted to conventional mechanical ventilation. LPA was initiated at once, resulting in an immediate increase in heart rate ($p = 0.0002$), cardiac output ($p = 0.0002$), oxygen delivery (D_{O_2}, $p = 0.0003$), and mixed venous P_{O_2} ($p = 0.0006$), with a maintained systemic oxygen consumption ($p = 0.52$). The mean pulmonary arterial pressure markedly increased (mean increment 8.8 mm Hg; $p < 0.0001$), but the pulmonary vascular resistance did not change ($p = 0.32$). Cardiac filling pressures increased ($p < 0.001$) and the systemic vascular resistance fell ($p = 0.003$). All these alterations were progressively attenuated in the course of the first 36 h, despite persisting hypercapnia. Plasma lactate suffered a progressive decrement along the early period in LPA but not in control patients ($p < 0.0001$). No hemodynamic consequences of LPA were noticed in the late period and renal function was preserved. A multivariate analysis suggested that these acute hyperdynamic effects were related to respiratory acidosis, with no depressant effects ascribed to high PEEP levels. In contrast, high plateau pressures were associated with cardiovascular depression. Thus, as long as sufficiently low distending pressures are concomitantly applied, the sudden installation of PHY plus $PEEP_{IDEAL}$ induces a transitory hyperdynamic state and pulmonary hypertension without harmful consequences to this young ARDS population.

Increased Rate of Fractures and Severe Kyphosis: Sequelae of Living into Adulthood with Cystic Fibrosis—Aris RM, Renner JB, Winders AD, Buell HE, Riggs DB, Lester GE, Ontjes DA. Ann Intern Med 1998;128(3):186.

BACKGROUND: Osteoporosis occurs in patients with cystic fibrosis as they age, but its clinical implications are not well defined. OBJECTIVE: To determine the clinical effect of decreased bone mineral density in adults with cystic fibrosis and to assess possible clinical predictors of osteoporosis. DESIGN: Retrospective cohort study. SETTING: Academic cystic fibrosis center. PATIENTS: 70 adults with late-stage cystic fibrosis who were referred for lung transplantation. MEASUREMENTS: Bone mineral density was measured with dual-energy x-ray absorptiometry, patient-reported fracture events were confirmed by radiography, and kyphosis angles were measured by using the Cobb method. RESULTS: Mean bone mineral densities for the spine, femur, and total body were severely depressed in patients with cystic fibrosis, averaging 2 SDs below those of age-matched normal controls ($p < 0.001$). Patient interviews showed that 54 fractures had occurred over 1,410 patient-years, and chest radiographs showed evidence of 14 additional rib and 62 additional vertebral compression fractures. The database (which covered 1,410 patient-years) showed that fracture rates were approximately twofold greater in women with cystic fibrosis aged 16 to 34 years ($p = 0.015$) and men with cystic fibrosis aged 25 to 45 years ($p = 0.04$) than in the general population. Vertebral compression and rib fractures were 100- and 10-fold more common than expected, respectively ($p < 0.001$ for both comparisons). The mean kyphosis angle (± SD) for this group was markedly abnormal (44 ± 14°; 62% ≥ 40°) and probably contributed to diminished stature (mean height loss, 5.8 cm in men with cystic fibrosis and 5.9 cm in women with cystic fibrosis). Cumulative prednisone dose, body mass index, and age at puberty were the strongest predictors of bone mineral density. CONCLUSIONS: Osteoporosis is universal in adults with late-stage cystic fibrosis, and its complications include increased fracture rates and severe kyphosis.

A Clinical Trial of Vena Caval Filters in the Prevention of Pulmonary Embolism in Patients with Proximal Deep-Vein Thrombosis. Prevention Du Risque d'Embolie Pulmonaire Par Interruption Cave Study Group—Decousus H, Leizorovicz A, Parent F, Page Y, Tardy B, Girard P, et al. N Engl J Med 1998;338(7):409.

BACKGROUND: The efficacy and safety of vena caval filters in the prevention of pulmonary embolism in patients with proximal deep-vein thrombosis is still a matter of debate. METHODS: Using a 2-by-2 factorial design, we randomly assigned 400 patients with proximal deep-vein thrombosis who were at risk for pulmonary embolism to receive a vena caval filter (200 patients) or no filter (200 patients), and to receive low-molecular-weight heparin (enoxaparin, 195 patients) or unfractionated heparin (205 patients). The rates of recurrent venous thromboembolism, death, and major bleeding were analyzed at Day 12 and at 2 years. RESULTS: At Day 12, 2 patients assigned to receive filters (1.1%), as compared with 9 patients assigned to receive no filters (4.8%), had symptomatic or asymptomatic pulmonary embolism (odds ratio, 0.22; 95% confidence interval [CI], 0.05 to 0.90). At 2 years, 37 patients assigned to the filter group (20.8%), as compared with 21 patients assigned to the no-filter group (11.6%), had recurrent deep-vein thrombosis (odds ratio, 1.87; 95% CI, 1.10 to 3.20). There were no significant differences in mortality or the other outcomes. At Day 12, 3 patients assigned to low-molecular-weight heparin (1.6%), as compared with 8 patients assigned to unfractionated heparin (4.2%), had symptomatic or asymptomatic pulmonary embolism (odds ratio, 0.38; 95% CI, 0.10 to 1.38). CONCLUSIONS: In high-risk patients with proximal deep-vein thrombosis, the initial beneficial effect of vena caval filters for the prevention of pulmonary embolism was counterbalanced by an excess of recurrent deep-vein thrombosis, without any difference in mortality. Our data also confirmed that low-molecular-weight heparin was as effective and safe as unfractionated heparin for the prevention of pulmonary embolism.

Diagnosis of Pulmonary Tuberculosis Using PCR Assays on Sputum Collected within 24 Hours of Hospital Admission—Cohen RA, Muzaffar S, Schwartz D, Bashir S, Luke S, McGartland LP, Kaul K. Am J Respir Crit Care Med 1998;157(1):156.

There have been few studies evaluating the efficacy of polymerase chain reaction (PCR) testing in front-line clinical practice. We assessed the diagnostic yield of PCR prospectively in a blinded study of patients admitted to rule out tuberculosis and compared PCR results to a culture and clinical diagnosis of tuberculosis. Specimens were sent for routine smear, culture, and PCR analysis. Sputum sediments were submitted for PCR amplification of IS6110 sequences by an in-house assay and also the Roche Amplicor PCR assay targeting 16s ribosomal RNA genes. Eighty-five patients were enrolled: 27 patients had cultures positive for tuberculosis; 12 were smear-positive. PCR by both assays on the first specimen picked up all patients smear-positive on any specimen. A positive PCR on at least 1 of 2 specimens collected in the first 24 hours was 85 and 74% sensitive and 88 and 93% specific for tuberculosis by the in-house and Roche techniques, respectively. Sensitivity in smear-negative patients was 73 and 53%, respectively. The in-house PCR detected 100% and Roche detected 95% of patients with more than paucibacillary (> 20 colonies) tuberculosis. We conclude that PCR may be a useful tool to evaluate patients for tuberculosis within the first hospital day.

Risk of Fatal Pulmonary Embolism in Patients with Treated Venous Thromboembolism—Douketis JD, Kearon C, Bates S, Duku EK, Ginsberg JS. JAMA 1998;279(6):458.

CONTEXT: The most serious complication of deep vein thrombosis (DVT) or nonfatal pulmonary embolism (PE) is fatal PE. However, reliable estimates as to the risk of fatal PE in patients with treated DVT or PE are lacking. OBJECTIVE: To provide reliable estimates of the risk of fatal PE and the case-fatality rate of recurrent DVT or PE among patients presenting with symptomatic DVT or PE, during and following 3 months of anticoagulant therapy. DATA SOURCES: A MEDLINE literature search was performed to iden-

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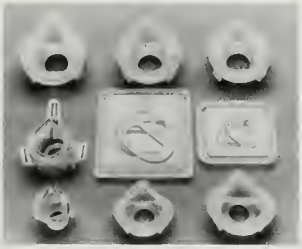

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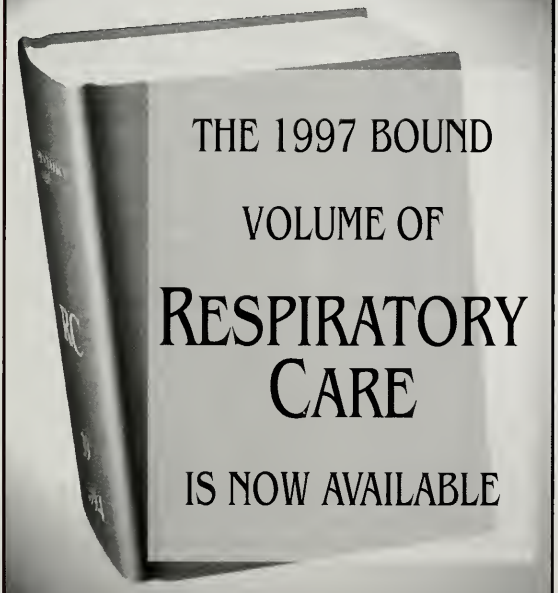

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tify prospective studies in which patients with symptomatic DVT or PE were treated with 5 to 10 days of heparin and 3 months of oral anticoagulants. We searched the years 1966 to September 1997 using the search terms thrombophlebitis, diagnosis, drug therapy, and prognosis. Current contents and bibliographies were also scanned. DATA EXTRACTION: Of 137 retrieved studies, 25 studies satisfied predetermined methodologic criteria and were included in the analysis. DATA SYNTHESIS: Among patients presenting with DVT, the rate of fatal PE during anticoagulant therapy was 0.4% (95% confidence interval [CI], 0.2%-0.6%); following anticoagulant therapy it was 0.3/100 patient-years (95% CI, 0.1-0.8). The case-fatality rate of recurrent DVT or PE during anticoagulant therapy was 8.8% (95% CI, 5.0%-14.1%); following anticoagulant therapy it was 5.1% (95% CI, 1.4%-12.5%). Among patients presenting with PE, the rate of fatal PE during anticoagulant therapy was 1.5% (95% CI, 0.9%-2.2%); following anticoagulant therapy it was 0/265 patient-years (95% CI, 0-3.6). The case-fatality rate of recurrent DVT or PE among patients presenting with PE was 26.4% (95% CI, 16.7%-38.1%). CONCLUSION: Among patients with symptomatic PE or DVT who are treated with anticoagulants for 3 months, fatal PE is rare during and following anticoagulant therapy. Patients presenting with PE are more likely to die of recurrent PE or DVT than are patients presenting with DVT.

Dyspnea Ratings for Prescription of Cross-Modal Exercise in Patients with COPD—Horowitz MB, Mahler DA. Chest 1998;113(1):60.

OBJECTIVE: To investigate the ability of patients with chronic obstructive pulmonary disease (COPD) to reproduce an exercise intensity accurately on the treadmill using dyspnea ratings obtained during incremental exercise on the cycle ergometer (cross-modal exercise prescription). DESIGN: Five visits over an 8-week period. PATIENTS: Thirteen symptomatic patients with stable COPD. Age was 67 ± 6 years (mean ± SD). FEV_1 was 1.15 ± 0.22 L (45 ± 7% predicted). INTERVENTIONS: At each visit, patients performed spirometry and exercise. Visit 1 was a practice incremental exercise test on the cycle ergometer. At Visit 2 (1 week later), patients estimated the intensity of dyspnea using the 0 to 10 category-ratio scale during an incremental exercise test on the cycle ergometer (cycle estimation trial). Visit 3, 5 weeks later, was a practice session on the treadmill. At Visit 4, 1 week later, patients were instructed to produce specific intensities of dyspnea (ie, dyspnea targets) at 50% and at anaerobic threshold (AT) or 80% of peak oxygen consumption ($\dot{V}_{O_2}$) as calculated from results at Visit 2 (treadmill production trial). Visit 5, 1 week later, was the treadmill estimation trial. MEASUREMENTS & RESULTS: Lung function was stable at all visits. Dyspnea ratings were 1.9 ± 0.9 at 50% of $\dot{V}_{O_2}$ and 5.6 ± 1.5 at AT/80% of peak $\dot{V}_{O_2}$ (17.5 ± 3.3 mL/kg/min). The $\dot{V}_{O_2}$ at the treadmill production trial (761 ± 185 mL/min) was significantly higher than at the cycle estimation trial (612 ± 159 mL/min) at the low dyspnea target ($p < 0.0002$; upward bias, 26 ± 16%). In contrast, there was no significant difference in $\dot{V}_{O_2}$ values (929 ± 176 mL/min vs 948 ± 259 mL/min) at the high dyspnea target ($p > 0.5$; 0 ± 11% bias). CONCLUSIONS: Patients with COPD can use dyspnea ratings from an incremental cycle ergometry test to regulate exercise on the treadmill without systematic bias at an intensity of 80% of peak $\dot{V}_{O_2}$, but exceed the desired $\dot{V}_{O_2}$ when using the dyspnea rating at an intensity of 50% of peak $\dot{V}_{O_2}$.

Depressed Mood and Survival in Seriously Ill Hospitalized Adults. The SUPPORT Investigators—Roach MJ, Connors AF, Dawson NV, Wenger NS, Wu AW, Tsevat J, et al. Arch Intern Med 1998;158(4):397.

OBJECTIVES: To assess the relationship among depressed mood, physical functioning, and severity of illness and to determine the relationship between depressed mood and survival time, controlling for severity of illness, baseline functioning, and characteristics of patients. METHODS: Prospective cohort study of data for 3,529 seriously ill hospitalized adults who received care at 5 tertiary care teaching hospitals and who completed a depressed mood assessment 7 to 11 days after admission to the study. The Profile of Mood States depression subscale was used to assess depressed mood. A stratified Cox proportional hazards model was used to assess the independent effect of depressed mood on survival time, adjusting for demographic characteristics of patients and health status. RESULTS: Greater magnitudes of depressed mood were associated with worse levels of physical functioning ($r = 0.151$; $p < 0.001$) and more severity of illness. Depressed mood was associated with reduced survival time after adjusting for patient demographics and health status (hazards ratio, 1.134; 95% confidence interval, 1.071-1.200; $p \leq 0.001$). CONCLUSION: Seriously ill patients should be assessed for the presence of depressed mood even if they have not been given a diagnosis of depression. Further study is needed to determine whether interventions aimed at relieving depressed mood may improve prognosis.

Objective Estimates of the Probability of Death from Burn Injuries—Ryan CM, Schoenfeld DA, Thorpe WP, Sheridan RL, Cassem EH, Tompkins RG. N Engl J Med 1998;338(6):362.

BACKGROUND: Over the past 20 years, there has been remarkable improvement in the chances of survival of patients treated in burn centers. A simple, accurate system for objectively estimating the probability of death would be useful in counseling patients and making medical decisions. METHODS: We conducted a retrospective review of all 1,665 patients with acute burn injuries admitted from 1990 to 1994 to Massachusetts General Hospital and the Shriners Burns Institute in Boston. Using logistic-regression analysis, we developed probability estimates for the prediction of mortality based on a minimal set of well-defined variables. The resulting mortality formula was used to determine whether changes in mortality have occurred since 1984, and it was tested prospectively on all 530 patients with acute burn injuries admitted in 1995 or 1996. RESULTS: Of the 1,665 patients (mean [± SD] age, 21 ± 20 years; mean burn size, 14 ± 20% of body-surface area), 1,598 (96%) lived to discharge. The mean length of stay was 21 ± 29 days. Three risk factors for death were identified: age > 60 years, more than 40% of body-surface area burned, and inhalation injury. The mortality formula we developed predicts 0.3%, 3%, 33%, or approximately 90% mortality, depending on whether 0, 1, 2, or 3 risk factors are present. The results of the prospective test of the formula were similar. A large increase in the proportion of patients who chose not to be resuscitated complicated comparisons of mortality over time. CONCLUSIONS: The probability of mortality after burns is low and can be predicted soon after injury on the basis of simple, objective clinical criteria.

Reduction in Pulmonary Vascular Resistance with Long-Term Epoprostenol (Prostacyclin) Therapy in Primary Pulmonary Hypertension—McLaughlin VV, Genthner DE, Panella MM, Rich S. N Engl J Med 1998;338(5):273.

BACKGROUND: Primary (idiopathic) pulmonary hypertension is a progressive, fatal disease. Conventional therapy with anticoagulant and vasodilator drugs may improve symptoms and survival among selected patients, but there is no evidence that the disease can be reversed. METHODS: We evaluated the effects of long-term therapy (ie, for more than 1 year) with intravenous epoprostenol (prostacyclin) in patients with advanced primary pulmonary hypertension. The baseline evaluation included an assessment of pulmonary vascular dilation in response to intravenous adenosine. The epoprostenol dose was increased monthly to the maximum tolerated. Long-term therapy was evaluated by measuring improvement in symptoms, exercise capacity, and hemodynamic measures. RESULTS: We evaluated 27 patients with primary pulmonary hypertension over a mean (± SD) period of 16.7 ± 5.2 months. Intravenous adenosine had a variable effect on pulmonary vascular resistance (mean reduction, 27%; range, 0 to 56; $p < 0.001$). Epoprostenol therapy was initiated and the rate of infusion was increased by an average of 2.4 ng/kg of body weight per minute each month. Twenty-six of the 27 patients had improvement in symptoms and hemodynamic measures, and overall, pulmonary vascular resistance declined by 53% to 7.9 ± 3.8 resistance units ($p < 0.001$)

at the time of restudy. The long-term effects of epoprostenol exceeded the short-term pulmonary vasodilator response to adenosine in all but 1 patient. Seven of the 8 patients who had minimal pulmonary vasodilation in response to adenosine (mean reduction in resistance units, < 20%) still had a significant reduction in pulmonary vascular resistance when treated with epoprostenol (mean, 39 ± 14%; p = 0.002). CONCLUSIONS: In primary pulmonary hypertension, long-term therapy with epoprostenol lowers pulmonary vascular resistance beyond the level achieved in the short term with intravenous adenosine. Epoprostenol appears to have sustained efficacy in this disorder.

Evaluation of a Ventilation Strategy To Prevent Barotrauma in Patients at High Risk for Acute Respiratory Distress Syndrome. Pressure- and Volume-Limited Ventilation Strategy Group—Stewart TE, Meade MO, Cook DJ, Granton JT, Hodder RV, Lapinsky SE, et al. N Engl J Med 1998;338(6):355.

BACKGROUND: A strategy of mechanical ventilation that limits airway pressure (P_{aw}) and tidal volume (V_T) while permitting hypercapnia has been recommended for patients with acute respiratory distress syndrome (ARDS). The goal is to reduce lung injury due to overdistention. However, the efficacy of this approach has not been established. METHODS: Within 24 hours of intubation, patients at high risk for ARDS were randomly assigned to either pressure- and volume-limited ventilation (limited-ventilation group), with the peak inspiratory pressure (P_{Imax}) maintained at ≤ 30 cm H_2O and V_T at ≤ 8 mL/kg of body weight, or to conventional ventilation (control group), with the P_{Imax} allowed to rise as high as 50 cm H_2O and V_T at 10-15 mL/kg. All other ventilatory variables were similar in the 2 groups. RESULTS: A total of 120 patients with similar clinical features underwent randomization (60 in each group). The patients in the limited-ventilation and control groups were exposed to different mean (± SD) V_T (7.2 ± 0.8 vs 10.8 ± 1.0 mL/kg, respectively; p < 0.001) and P_{Imax} (23.6 ± 5.8 vs 34.0 ± 11.0 cm H_2O, p < 0.001). Mortality was 50% in the limited-ventilation group and 47% in the control group (relative risk, 1.07; 95% confidence interval, 0.72 to 1.57; p = 0.72). In the limited-ventilation group, permissive hypercapnia (P_{aCO_2} > 50 mm Hg) was more common (52% vs 28%, p = 0.009), more marked (54.4 ± 18.8 vs 45.7 ± 9.8 mm Hg, p = 0.002), and more prolonged (146 ± 265 vs 25 ± 22 hours, p = 0.017) than in the control group. The incidence of barotrauma, the highest multiple-organ-dysfunction score, and the number of episodes of organ failure were similar in the 2 groups; however, the numbers of patients who required paralytic agents (23 vs 13, p = 0.05) and dialysis for renal failure (13 vs 5, p = 0.04) were greater in the limited-ventilation group than in the control group. CONCLUSIONS: In patients at high risk for ARDS, a strategy of mechanical ventilation that limits P_{Imax} and V_T does not appear to reduce mortality and may increase morbidity.

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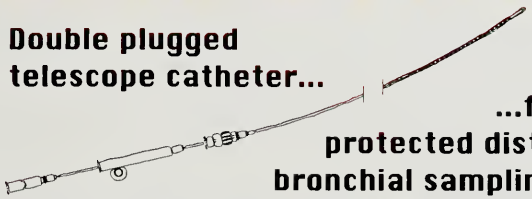

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Contribution of the Endotracheal Tube and The Upper Airway to Breathing Workload—Straus C, Louis B, Isabey D, Lemaire F, Harf A, Brochard L. Am J Respir Crit Care Med 1998; 157(1):23.

The influence of the endotracheal tube (ETT) during a T-piece trial remains controversial. Our aim was to compare the work of breathing of 14 successfully extubated patients at the end of a 2-hour trial (T) and after extubation (E) of the trachea, and to assess, using the acoustic reflection method, the resistance of the endotracheal tube and of the supraglottic airway as well as their related work. We found that the work of breathing of the patients was identical between T and E (1.72 ± 0.59 vs 1.63 ± 0.45 J/L; p = 0.50 and 23.5 ± 10.6 vs 22.6 ± 9.7 J/min; p = 0.70). There was no significant difference between the beginning and the end of the T-piece trial (1.57 ± 0.53 vs 1.72 ± 0.59 J/ L, p = 0.10). The work caused by the ETT amounted to 11.0 ± 3.9% of the total work of breathing. The supraglottic airway resistance was in the normal range and was significantly smaller than the endotracheal tube resistance (0.79 ± 0.4 vs 1.43 ± 0.31 cm H_2O M s/L; p = 0.008, flow = 0.25 L/s). We conclude that a 2-hour trial of spontaneous breathing through an endotracheal tube well mimics the work of breathing performed after extubation, in patients who pass a weaning trial and do not require reintubation.

COPD and Other Diseases in Chronically Ventilated Patients in a Prolonged Respiratory Care Unit: A Retrospective 20-Year Survival Study—Votto J, Brancifort JM, Scalise PJ, Wollschlager CM, ZuWallack RL. Chest 1998;113(1):86.

OBJECTIVES: To evaluate the long-term prognosis of ventilator-dependent patients. DESIGN: Retrospective study. SETTING: A prolonged respiratory care unit (PRCU). The PRCU provides comprehensive medical, nursing, and respiratory care to tracheostomized, ventilator-dependent adult patients who had failed all attempts at weaning. Because of their medical complexity, these patients could not be discharged to a lower level of care. PATIENTS: Of the 293 patients admitted to the

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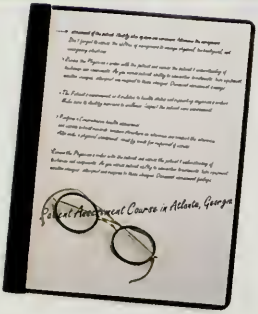

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- C. Understand the principle of patient self-determination
- D. Appreciate the psychological, social, and physical characteristics of wellness/illness
- E. Recognize the patient's right to confidentiality
- F. List the principles of informed consent

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- A. Review patient records
- B. Structure an interview
- C. Conduct an interview
- D. Conduct a physical assessment
- E. Identify needs for referral
- F. Identify patients with potential for high-risk medical complications
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- A. Identify sites of care and caregivers
- B. Interview the caregivers
- C. Assess the abilities of caregivers to manage physical, technological, and emergency situations
- D. Assess home health resources

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- A. Review the physician's order with the patient
- B. Assess the patient's understanding of technique and components

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C. Assess patient ability to administer treatments, use equipment, monitor changes, interpret and respond to those changes
D. Document assessment findings

5. Assess the Patient's Environment as It Relates to Health Status and Supporting Physician's Orders
A. Identify barriers to wellness

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B. Inspect the patient care environment
C. Determine the resources available to the patient and family
D. Document assessment findings

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6. Assess Continued Appropriateness of Physician Orders and Care Plan
A. Review physician orders
B. Evaluate the patient's response to physician orders
C. Evaluate the appropriateness of the orders and the care plan
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E. Document assessment findings

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PRCU over a 20-year period beginning January 1, 1977, 145 had respiratory failure from COPD, 22 from spinal cord disease or trauma, 34 from primary CNS disease, 50 from primary neuromuscular disease, and 16 from chest wall disease. Twenty-six patients were not classifiable into the above categories. MEASUREMENTS: Demographics, diagnoses, and survival data were reviewed. The survival of patients with COPD was compared with the other diagnosis categories using the Cox proportional hazards model. RESULTS: The median survival for the entire group was 9 months; younger age and female gender were both predictive of longer survival (both, $p < 0.001$). The median survival of those with COPD (5 months) was significantly shorter than that of patients with spinal cord disease (47 months), neuromuscular disease (17 months), and chest wall disease (27 months) (all, $p < 0.01$). These differences in survival were present even with inclusion of gender and age in the model as covariates. The survival of patients with CNS disease was not significantly different from survival of patients with COPD. CONCLUSION: Chronically ventilated patients with respiratory failure from COPD have a significantly worse prognosis than patients with respiratory failure from other causes.

A Randomized Controlled Trial Comparing Zileuton with Theophylline in Moderate Asthma. The Zileuton Study Group—Schwartz HJ, Petty T, Dube LM, Swanson LJ, Lancaster JF. Arch Intern Med 1998;158(2):141.

BACKGROUND: Zileuton, a leukotriene pathway inhibitor, was compared with slowly absorbed theophylline in a randomized, double-blind study of patients with chronic asthma. The primary efficacy measure was improvement in forced expiratory volume in 1 second (FEV_1). METHODS: Eligibility criteria included FEV_1 of 40% to 80% of predicted, documented reversibility of airway disease, and age 18 to 60 years. Initially, the theophylline dosage was titrated to achieve trough concentrations of 8 to 15 μg/mL. After washout and 1-week placebo lead-in, patients were randomly assigned to 13 weeks of the appropriate theophylline dose or zileuton, 400 or 600 mg 4 times daily. The FEV_1 was measured before the morning dose at 2-week intervals and serially after the dose on Days 36 and 92. Patients kept daily diaries of asthma symptoms, β-agonist usage, and peak expiratory flow rate; on Days 36 and 92, they completed quality-of-life questionnaires. RESULTS: Of 471 eligible patients at 38 centers, 377 were randomly assigned to the study; 313 completed the study. On first-dose administration, all groups showed 11% to 13% improvement in FEV_1 within 30 minutes. Patients who received zileuton, 400 mg, had significantly greater improvement at several points than did theophylline-treated patients. The range of long-term maximum improvement in FEV_1 in the groups was 30% to 34% ($p = 0.40$ for zileuton 600 mg; $p = 0.90$ for zileuton 400 mg vs theophylline). Initially, the theophylline group improved significantly more in symptom scores, β-agonist usage, and peak expiratory flow rate, but at maximal effect there was no significant difference. All groups showed significant improvement in quality of life. No overall differences were observed between the zileuton dosage groups. Adverse events were comparable in all groups. CONCLUSION: Zileuton appears as effective and safe as theophylline in patients with chronic asthma.

Pulmonary Infiltrates, Eosinophilia, and Cardiomyopathy following Corticosteroid Withdrawal in Patients with Asthma Receiving Zafirlukast—Wechsler ME, Garpestad E, Flier SR, Kocher O, Weiland DA, Polito AJ, et al. JAMA 1998;279(6):455.

CONTEXT: Zafirlukast is a potent leukotriene antagonist that recently was approved for the treatment of asthma. As use of this drug increases, adverse events that occur at low frequency or in populations not studied in premarketing clinical trials may become evident. OBJECTIVE: To describe a clinical syndrome associated with zafirlukast therapy. DESIGN: Case series. PATIENTS: Eight adults (7 women and 1 man) with steroid-dependent asthma who received zafirlukast. MAIN OUTCOME MEASURES: Development of a clinical syndrome characterized by pulmonary infiltrates, cardiomyopathy, and eosinophilia following the withdrawal of corticosteroid treatment. RESULTS: The clinical syndrome developed while patients were receiving zafirlukast from 3 days to 4 months and from 3 days to 3 months after corticosteroid withdrawal. All 8 patients developed leukocytosis (range, 14.5-27.6 $\times 10^9$/L) with eosinophilia (range, 0.19-0.71). Six patients had fever (temperature > 38.5° C), 7 had muscle pain, 6 had sinusitis, and 6 had biopsy evidence of eosinophilic tissue infiltration. The clinical syndrome improved with discontinuation of zafirlukast treatment and reinitiation of corticosteroid treatment or addition of cyclophosphamide treatment. COMMENT: Development of pulmonary infiltrates, cardiomyopathy, and eosinophilia may have occurred independent of zafirlukast use or may have resulted from an allergic response to this medication. We suspect that these patients may have had a primary eosinophilic infiltrative disorder that had been clinically recognized as asthma, was quelled by steroid treatment, and was unmasked following corticosteroid withdrawal facilitated by zafirlukast.

The Relation of Pneumothorax and Other Air Leaks to Mortality in the Acute Respiratory Distress Syndrome—Weg JG, Anzueto A, Balk RA, Wiedemann HP, Pattishall EN, Schork MA, Wagner LA. N Engl J Med 1998;338(6):341.

BACKGROUND: In patients with the acute respiratory distress syndrome (ARDS), pneumothorax and other air leaks—any extrusion of air outside the tracheobronchial tree—have been attributed to high ventilatory pressures or volumes and linked to increased mortality. METHODS: We analyzed data from a prospective trial of aerosolized synthetic surfactant in 725 patients with ARDS induced by sepsis. We compared the ventilatory pressures and volumes in the patients without any air leaks (the highest values during the 5-day study) with the pressures and volumes in those with pneumothorax or with any air leaks (the highest values during the 16- and 24-hour periods before the complication developed). RESULTS: Fifty patients (6.9%) had pneumothorax, and 77 (10.6%) had pneumothorax or other air leaks. There were no significant differences between patients with air leaks and those without air leaks in any pressure or volume examined. Overall mortality at 30 days was 40.0% (95% confidence interval [CI], 36.4 to 43.6); among the patients with pneumothorax, it was 46.0% (95% CI, 32.2 to 59.8), and among those without pneumothorax, it was 39.3% (95% CI, 35.6 to 43.0; $p = 0.35$). The mortality rate was 45.5% (95% CI, 34.4 to 56.6) in the group with any air leaks and 39.0% (95% CI, 35.3 to 42.8) in the group without air leaks ($p = 0.28$). CONCLUSIONS: In patients with sepsis-induced ARDS who were receiving mechanical ventilation with conventional pressures and volumes, there were no significant correlations between high ventilatory pressures or volumes and the development of pneumothorax or other air leaks. Pneumothorax or other air leaks were not associated with a significantly increased mortality rate.

Lung Transplantation in Cystic Fibrosis: Consensus Conference Statement—Yankaskas JR, Mallory GB Jr. Chest 1998;113(1):217.

The first successful heart-lung and lung transplant operations in cystic fibrosis (CF) patients were performed in 1983 and 1987, respectively. Lung transplantation is now available at dozens of centers in North America, Europe, and Australia. Recent technical developments and the major limitations of donor organ availability prompted the CF Foundation to sponsor a meeting of 37 experts to evaluate the state of the art in lung transplantation for CF, highlighting areas of consensus, practice variations, and controversy. This document summarizes the work of that group.

Effects of Long-Term Oxygen Therapy on Pulmonary Hemodynamics in COPD Patients: A 6-Year Prospective Study—Zielinski J, Tobiasz M, Hawrylkiewicz I, Sliwinski P, Palasiewicz G. Chest 1998;113(1):65.

OBJECTIVE: To investigate effects of 6 years of domiciliary oxygen therapy on pulmonary hemodynamics in a large group of COPD patients. DESIGN: Prospective longitudinal study with serial measurements. SETTING: Research insti-

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tute of pulmonary diseases. PATIENTS: Ninety-five patients (72 men, 23 women), mean age 58 ± 9 years, had COPD but were free of any other serious disease. Functional characteristics at entry, mean ± SD, were as follows: FVC = 2.24 ± 0.51 L; FEV_1 = 0.84 ± 0.31 L; P_{aO_2} = 55 ± 6 mm Hg; P_{aCO_2} = 48 ± 9 mm Hg; mean pulmonary arterial pressure (PAP) = 28 ± 11 mm Hg; and pulmonary vascular resistance (PVR) = 353 ± 172 dyne × s × cm^{-5}. METHODS: Pulmonary hemodynamics were investigated using Swan-Ganz thermodilution catheters. After initial assessment, all patients were started on a regimen of long-term oxygen therapy (LTOT). Follow-up consisted of medical examination, spirometry, and arterial blood gas analysis every 3 months. Pulmonary artery catheterization was repeated every 2 years. RESULTS: Seventy-three subjects survived 2 years of LTOT. In 39 subjects catheterized after 2 years, PAP fell from 25 ± 8 to 23 ± 6 mm Hg (not significant [NS]). From 31 patients who completed 4 years of LTOT, hemodynamic data were obtained in 20. In these 20 patients, PAP averaged 24 ± 7 mm Hg at entry, and 23 ± 5 and 26 ± 6 mm Hg after 2 and 4 years, respectively (NS). In 12 patients who completed 6 years of LTOT, PAP was 25 ± 7 at entry, and 21 ± 4, 26 ± 7, and 26 ± 6 mm Hg at 2, 4, and 6 years, respectively ($p < 0.01$ for 2 vs 6 years). PVR was 313 ± 159 dyne × s × cm^{-5} at entry, and 268 ± 110, 344 ± 82, and 332 ± 205 dyne × s × cm^{-5} at 2, 4, and 6 years, respectively (NS). During 6 years of follow-up, P_{aO_2} decreased from 61 ± 3 to 46 ± 9 mm Hg ($p < 0.001$) and P_{aCO_2} increased from 44 ± 13 to 49 ± 9 mm Hg ($p < 0.01$). CONCLUSION: LTOT for 14 to 15 h/d resulted in a small reduction in pulmonary hypertension after the first 2 years followed by a return to initial values and subsequent stabilization of PAP over 6 years. The long-term stabilization of pulmonary hypertension occurred despite progression of the airflow limitation and of hypoxemia.

Influence of Tidal Volume on the Distribution of Gas between the Lungs and Stomach in the Nonintubated Patient Receiving Positive-Pressure Ventilation—Wenzel V, Idris AH, Banner MJ, Kubilis PS, Williams JL Jr. Crit Care Med 1998;26(2):364.

OBJECTIVES: When ventilating a nonintubated patient in cardiac arrest, the European Resuscitation Council has recently recommended a decrease in the tidal volume from 0.8 to 1.2 L to 0.5 L, partly in an effort to decrease peak flow rate, and therefore, to minimize stomach inflation. The purpose of the present study was to examine the validity of the European Resuscitation Council's recommendation in terms of gas distribution between lungs and stomach in a bench model that simulates ventilation of a nonintubated patient with a self-inflatable bag representing tidal volumes (V_T) of 0.5 and 0.75 L. DESIGN: A bench model of a patient with a nonintubated airway was used consisting of face mask, mannequin head, training lung (lung compliance, 50 mL/cm H_2O; airway resistance, 5 cm H_2O/L/sec), adjustable lower esophageal sphincter pressure (LESP) and simulated stomach. SETTING: University hospital laboratory. SUBJECTS: Thirty health care professionals. INTERVENTIONS: Health care professionals performed 1-min bag-mask ventilation at each LESP level of 5, 10, and 15 cm H_2O at a rate of 12 breaths/min, using an adult and pediatric self-inflating bag, respectively. Volunteers were blinded to the LESP, which was randomly varied. MEASUREMENTS & RESULTS: Both types of self-inflating bags induced stomach inflation, with higher stomach and lower lung V_T when the LESP was decreased. Lung V_T with the pediatric bag was significantly ($p < 0.05$) lower at all LESP levels when compared with the adult bag, and ranged between 240 mL at an LESP of 15 cm H_2O and 120 mL at an LESP of 5 cm H_2O. Stomach V_T with the adult bag ranged between 250 mL at an LESP of 15 cm H_2O and increased to 550 mL at an LESP of 5 cm H_2O. Stomach V_T with the pediatric bag was significantly lower ($p < 0.05$) at all LESP levels when compared with the adult bag and ranged between 70 mL at an LESP of 15 cm H_2O and 300 mL at an LESP of 5 cm H_2O. CONCLUSIONS: Our data support the recommendation of the European Resuscitation Council to decrease V_T to 0.5 L when ventilating a cardiac arrest victim with an unprotected airway. A small V_T may be a better trade-off in the basic life support phase, as this may provide reasonable ventilation while avoiding massive stomach inflation.

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SLEEP-DISORDERED BREATHING

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Non-Surgical Alternatives to Continuous Positive Airway Pressure

Lawrence J Epstein MD

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Summary

Introduction

Nasal continuous positive airway pressure (CPAP) has become the first-line treatment for patients with obstructive sleep apnea (OSA).[1] Use of CPAP results in immediate prevention of upper airway collapse and leads to correction of blood gas derangements, improved sleep continuity, and resolution of sleepiness symptoms and cognitive impairment.[2] The effectiveness of CPAP is limited by patient compliance because it is only effective when the patient wears the device. Initial subjective reports suggested high patient compliance, with 1-year compliance values at about 75%.[3] However, objective measurements, utilizing hour meters on the devices to measure actual use, showed much poorer compliance. Kribbs et al[4] found that only 46% of patients used their CPAP for at least 4 hours, 5 nights a week. Attempts to improve compliance with bi-level positive airway pressure,[4] positive reinforcement,[5] or desensitization therapy[6] have not been successful. As a result, investigators have been looking for alternative methods for treating OSA. Many surgical procedures have been developed and these are addressed in another paper in this issue. In this report, I focus on non-surgical alternatives, which can be classified into three categories: behavioral methods, medical therapies, and mechanical devices. Behavioral methods are predicated on the theory that some change in the patient's behavior will result in a reduction or elimination of OSA. Medical therapies involve administration of pharmacologic agents to directly prevent airway collapse, ameliorate the consequences of airway collapse, or treat an underlying medical problem that contributes to the development of OSA. Several mechanical devices other than CPAP have been developed in an attempt to maintain upper airway patency, and these devices constitute the third category.

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A version of this paper was presented by Dr Epstein during the RESPIRATORY CARE Journal Conference on Sleep-Disordered Breathing, held in Cancún, Mexico, October 17-19, 1997.

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Behavioral Alternatives

Weight Loss

There is a strong association between the development of OSA and the presence of obesity. In one clinic population of more than 1,000 OSA patients, two thirds were overweight (defined as > 20% above ideal body weight).[7] A cross-sectional epidemiologic study of thirty 60-year-old subjects, found that a 1-standard-deviation increase in body mass index (BMI) was associated with a fourfold increase in risk for OSA.[8] The mechanism responsible for this association is most likely related to the effects of obesity on upper airway structure and function.

Obesity could lead to narrowing of the upper airway either through deposition of fatty tissue in the pharyngeal walls or through the effect of mass loading on the pharynx by subcutaneous fat. Several studies using computed tomography (CT) have demonstrated that the upper airway of awake patients with OSA is narrower than those of controls.[9,10] Magnetic resonance imaging (MRI) has shown that fatty tissue is deposited in the lateral pharyngeal walls of OSA patients resulting in thickening of the lateral walls and narrowing of the pharyngeal lumen.[11] Davies and Stradling[12] found that neck circumference was a better predictor of the presence of OSA than measures of general obesity, such as the BMI, highlighting that obesity predisposes to the development of OSA by causing narrowing of the upper airway. Obesity-induced hypoxemia and changes in lung volume also may promote collapse of the upper airway.[13,14] During wakefulness, augmentation of the dilator action of the pharyngeal muscles maintains patency of the upper airway.[15] With the onset of sleep this neuromuscular compensation is lost, the airway collapses, and obstructive apnea occurs.

Given the significant contribution of obesity to the development of OSA, weight loss would be expected to be a successful treatment. Massive weight loss in morbidly obese patients dramatically improves apnea and gas exchange abnormalities.[16] Even relatively small weight losses can lead to significant reductions in apneic events and sleep fragmentation and improvement in oxygenation and daytime alertness.[17,18] Weight loss by any method can be beneficial in treating OSA. Reduction in OSA frequency and consequences has been demonstrated after dietary weight loss[16,19] and after gastric reduction or bypass surgery.[20,21] Use of anorectic medications such as fenfluramine and phentermine may be beneficial in obese patients with OSA. However, the paucity of controlled trials and recent reports of primary pulmonary hypertension[22] and heart valve damage[23] in anorectic drug users argues against wide-scale use until more investigation has been done.

Weight loss induces changes in upper airway anatomy and function. Pharyngeal adipose tissue volume, measured by MRI, was reduced after weight loss in patients who had a reduction in OSA.[24] Acoustic reflection evaluation of the upper airway of patients with reduced OSA following weight loss demonstrated a reduction in the lung volume dependence of pharyngeal area, indicating an improvement in pharyngeal wall and glottic function.[25]

Not all patients who lose weight have improvement in their OSA.[17,26] Other factors, such as craniofacial structure may cause anatomic narrowing of the upper airway and contribute to the development of OSA. The major limitations of weight loss as primary treatment for OSA are the length of time required to achieve effect, unpredictability of the magnitude of weight loss required to eliminate OSA, low success rate in maintaining long-term weight reduction, and health risks associated with weight cycling.[13,27] Despite these difficulties, weight loss should be considered in all overweight patients with OSA and, if successful, may be curative.

Position Training

Sleep position affects the occurrence and severity of OSA. Some patients have OSA only or predominantly in the supine position (as opposed to the lateral position).[28] The likelihood of position-dependent OSA is inversely correlated with body weight, with heavier patients tending not to have position-dependent OSA.[29] This position dependence is also affected by sleep stage, with up to 41% becoming nonposition dependent during rapid eye movement (REM) sleep.[30-32] A similar relationship has been found for the occurrence of OSA between the supine and upright positions.[33] The effect of sleep position on airway size and function is responsible for position-dependent OSA.

Body position is a determinant of upper airway size due to the consequences of gravity on the pharyngeal structures. The area of the pharynx decreases with the change from sitting upright to lying down in both apneic subjects and controls.[34-36] Why some OSA patients have differences in severity in the supine versus the lateral position is due to a combination of structural and functional changes. One study[37] found that while OSA patients had smaller upper airway areas than those who were not apneic, there was no difference between the lateral and supine pharyngeal cross-sectional areas of those with OSA. Pevernegie et al[38] similarly found no difference in mean cross-sectional area of the pharynx, measured by CT, between patients with positional and nonpositional OSA. However, there were differences in pharyngeal shape between the groups; the pharynx of patients with positional OSA tended to be more elliptical, collapsed more in the anteroposterior dimension, and had a smaller minimum cross-sectional area, all of which promote collapse in the supine position. Positioning also changes the functional capabilities of the pharyngeal muscles. Changing from an upright to a supine position increases the distensibility of the pharynx causing it to become floppier and more likely to collapse.[39] The stability of the airway is compromised to a greater degree in the supine than in the lateral position. The upper airway opening pressure (minimal CPAP required

to prevent collapse) is 50% less in the lateral position than the supine position.[40] The combination of a smaller pharyngeal airway with further position-dependent differences in shape and function in the supine position promotes airway collapse.

Investigators have evaluated methods for taking advantage of the position effects on airway collapse to treat patients with position-dependent OSA. Attempts to train people to stay off their backs take two forms: those that prevent rolling onto the back and those that detect when the person is on his back, causing arousal so he can roll off his back. The first category includes various kinds of restraints that prevent the patient from assuming the supine position, including foam wedges or sand bags placed against the sleeper's back, a pillow attached to the sleeper's back with a belt, or a fully stuffed backpack. The most common tactic in the second category is the use of a sock stuffed with a tennis ball and sown into the middle of the back of the sleeper's pajama top to cause awakening when supine. Position alarms that sense when the person is supine and emit a stimulus (eg, sound or vibration) to wake up the sleeper so he can turn from his back can also be used. Cartwright et al[41] demonstrated that the use of a gravity-activated position alarm effectively reduced the amount of time spent sleeping supine and, as a result, reduced the severity of the OSA, eliminating it in some cases. This effect was comparable to that achieved with a tongue-retaining oral appliance and could be sustained without the position alarm by about half the patients.[42] Although case reports indicate continued benefits of position training over several years, no study has documented the long-term effectiveness of position training.[43] Changing sleep posture is another form of position therapy. McEvoy et al[33] were able to reduce the frequency and severity of OSA in a subset of OSA patients by elevating the head of the bed to a 60° angle from the horizontal. Before using position training it is important to make sure that the patient has position-dependent OSA, that it persists during all stages of sleep, and that changing sleep position reduces OSA sufficiently.

Avoidance

A cardinal principle for patients with OSA is to avoid any agent or condition that would potentiate upper airway dilator muscle relaxation and promote collapse. Factors that have this action include alcohol, sedative-hypnotic and narcotic medications, cigarette smoking, and sleep deprivation.

Studies have shown that alcohol worsens apnea frequency, severity, and accompanying oxyhemoglobin desaturation in patients with OSA, and can induce apneas in nonapneic snorers and nonsnorers.[44,45] Alcohol selectively decreases neural input and electromyographic activity of pharyngeal dilator muscles while maintaining diaphragmatic activity.[46,47] This results in a decreased pharyngeal airspace, increased pharyngeal resistance and increased tendency to collapse.[48] The response to alcohol is greater in men than in either pre- or postmenopausal women.[49-51] The effects of alcohol are worse in the first few hours after ingestion,[52] so patients with OSA should be advised to refrain from drinking alcohol or at least not to have any for 4-5 hours prior to bedtime.

In a similar way, sedative agents can worsen OSA.[53,54] Benzodiazepines selectively depress pharyngeal dilator muscle activity[55,56] and can decrease the arousal response to hypercapnia and hypoxia.[57,58] This response may not occur in patients with mild OSA,[59] but individual response cannot be predicted in advance. As a result, sedative agents should be avoided in patients with OSA; if used, then carefully monitored.

A longitudinal epidemiologic study[60] of middle-aged adults showed that cigarette smoking was an independent risk factor for the presence of OSA. Current smokers have a greater risk than former or nonsmokers and heavy smokers have the greatest risk—almost 7 times that of nonsmokers. The mechanism of this increased risk is unknown. I believe that smoking cessation should be included in treatment programs for OSA.

Finally, all OSA patients need to be educated on the need to get adequate amounts of sleep. Sleep deprivation, independent of OSA, has been shown to change ventilatory patterns during sleep[61] and decrease the hypoxic and hypercapnic ventilatory responses, blunting ventilatory chemosensitivity.[62] In one study of OSA patients, the number and length of apneas increased after a single night of sleep deprivation.[63] Even if treated by other means, OSA patients should avoid sleep deprivation.

Medical Alternatives

Oxygen

One of the most common features of OSA is recurrent episodes of hypoxemia accompanying airway occlusion. In other disorders, such as chronic obstructive pulmonary disease (COPD), chronic hypoxemia has been associated with the development of pulmonary hypertension and right heart failure and can be corrected with the administration of supplemental oxygen.[64] OSA is also associated with the development of pulmonary hypertension and right heart failure; however, it is not clear whether the repetitive episodes of hypoxemia are responsible for these hemodynamic derangements. Other possibilities include the changes in sympathetic discharge accompanying the apnea-terminating arousal or the exaggerated swings in intrathoracic pressure seen during attempts to overcome airway obstruction. Regardless of the mechanism, one of the major goals for treating OSA is the elimination of hypoxemia.

The multiple effects of supplemental oxygen on events surrounding airway obstruction have been reviewed extensively by Fletcher and Munafo[65] and are summarized in Table 1. The use of oxygen decreases the nadir of oxygen desaturation and improves the overall saturation profile.[66] In addition, apnea duration is lengthened, leading to an increase in the P_{aCO_2} and

Table 1. Effects of Supplemental Oxygen Therapy in Obstructive Sleep Apneas*

Beneficial Effects	Adverse Effects	No Change
Improved saturation	Increased apnea duration	Hypersomnolence
Decreased apnea frequency	Increased P_{aCO_2}	
Decreased apnea time	Increased respiratory acidosis; changed mixed/central apneas to obstructive apneas	

*Based on information from Reference 65.

degree of respiratory acidosis accompanying apneas.[67] This change is most likely due to a reduction in the hypoxic ventilatory stimulus, which results in a delay in the arousal response.[68] Nonetheless, these events appear to be well tolerated because no increase in arrythmias or hemodynamic instability has been reported with oxygen use.[66,67,69] Several studies have shown a reduction in the number of hypopneas and a small reduction in number of apneas.[70-72] However, one study showed that, although overall apnea frequency was reduced and mixed and central apneas were markedly reduced with oxygen, the number of obstructive apneas increased, suggesting that low-flow oxygen shifts central and mixed apneas to obstructive apneas.[73] Despite the improvement in hypoxemia, studies of objective measures of sleepiness in patients with OSA show no improvement in hypersomnolence with supplemental oxygen.[71,73]

In summary, supplemental oxygen reduces the degree of hypoxemia accompanying OSA but does not substantially prevent airway collapse or sleep fragmentation. This is not surprising because supplemental oxygen does not address the central pathophysiologic event of OSA, which is pharyngeal muscle relaxation leading to collapse of an anatomically small airway. As a result, there is no improvement in daytime hypersomnolence. No information is available on the long-term cardiovascular effects of supplemental oxygen therapy—whether use of oxygen prevents pulmonary or systemic hypertension or right heart failure. Supplemental oxygen can be delivered safely to OSA patients via nasal cannula or transtracheal cannula.[74] At this time, oxygen cannot be considered a first-line therapy for OSA and should be considered only when more effective methods have failed and clinically important hypoxemia is present, as a temporary measure while awaiting institution of more effective therapy, or in patients with mild OSA without hypersomnolence.

Antidepressants

Interest in antidepressants as treatment for OSA started with recognition that some patients with narcolepsy had reductions in episodes of apnea following treatment with protriptyline. Other tricyclic antidepressants have sedating properties that limit their usefulness, whereas protriptyline is nonsedating. Protriptyline can produce a reduction in the frequency of apneas and degree of oxygen desaturation in some patients with OSA;[75,76] however, not all studies have found an apnea-suppressing effect.[77] Response tends to occur in patients with mild-to-moderate OSA but not in those with severe apnea.[78] The reduction may be due to protriptyline's suppressing effect on REM sleep,[79] the time during which the most severe apneas occur, or due to its ability to augment hypoglossal nerve activity and thus increase pharyngeal dilator muscle tone.[55] A high rate of anticholinergic side effects, such as dry mouth, urinary retention, impotency, and ataxia, limit the general use of protryptyline. One study[80] compared the effect of protriptyline with fluoxetine, a serotonin-uptake inhibitor. The response to fluoxetine was equivalent to that of protriptyline but was much better tolerated with fewer side effects.

The role of antidepressants in treating OSA is limited to patients with mild-to-moderate OSA who fail or are intolerant of more effective therapy. Fluoxetine appears to have equal efficacy to protriptyline with fewer side effects, but no data on long-term effectiveness are available.

Progesterone

Medroxyprogesterone is a respiratory stimulant that increases minute ventilation. Daily administration was effective in correcting awake blood gas abnormalities in patients with the obesity-hypoventilation syndrome.[81,82] Because OSA occurs frequently in patients with obesity-hypoventilation syndrome, it was hoped that a respiratory stimulant might be effective in OSA by increasing respiratory drive and improving upper airway muscle function. Early studies showed that a proportion of OSA patients treated with progesterone had a reduction in their apnea rates and improvement in oxygen desaturation.[83,84] However, many subsequent studies failed to find any therapeutic effect.[85-88] Collop et al[88] gave progesterone to men with OSA to see if it would provide protection against the effects of alcohol similar to that seen naturally in women. Progesterone improved oxygenation following alcohol ingestion, probably due to its stimulant effect, but had no impact on the frequency or length of obstructive apnea. Progesterone is not indicated in the treatment of OSA except as an adjunct in patients with a concurrent hypoventilation syndrome.

Other Agents

The ideal agent should selectively increase pharyngeal dilator muscle tone without affecting sleep architecture or causing clinically important side effects. Unfortunately, none of the additional agents investigated have had this profile. Other respiratory stimulants that have been studied but found to be unsuccessful in treating OSA include almitrine,[89] theophyl-

line,[90] and acetazolamide.[91,92] We have reported a series[93] that suggests some success treating postmenopausal women with OSA with sex hormones; however, one study of 15 women showed no success in reducing the severity of their OSA with these agents,[94] and this treatment cannot be recommended at this time. Use of nasal decongestants to reduce nasal resistance to airflow may reduce snoring but adds little to treatment of OSA.[26] Modafinil, a central nervous stimulant, decreased daytime sleepiness and increased vigilance but had no effect on obstructive events.[95] Additionally, naloxone, bromocriptine, and nicotine have been found to be ineffective in treating OSA.[96,97]

Correction of Other Medical Conditions

There is a relatively high prevalence of OSA in patients with hypothyroidism.[98] Several consequences of hypothyroidism could provide the mechanism for this association, including disease-associated weight gain, macroglossia, or skeletal muscle myopathy.[99] Thyroid hormone replacement reduces the number and severity of obstructive apneas, even before weight loss is achieved.[100] Clinicians should maintain a high level of suspicion for this disorder when evaluating OSA patients and check thyroid function tests when signs and symptoms suggest hypothyroidism.[101] Pharmacologic treatment of acromegaly with a somatostatin analog reverses upper airway anatomic abnormalities and can eliminate related OSA.[102] Any other neuromuscular disorder can predispose to OSA and should be considered when evaluating new patients. Human immunodeficiency virus disease can predispose to OSA by causing tonsillar hypertrophy as part of the generalized lymphadenopathy associated with the early stages of the disease.[103]

Mechanical Alternatives

Oral Appliances

The most successful alternatives to CPAP and surgery are the oral appliances (OA). These are mouthpieces that are worn while sleeping to prevent the airway from collapsing. There are currently over a dozen of these devices on the market. An extensive body of literature exists on the success of these devices. A full review, which is beyond the scope of this paper, can be found elsewhere.[104] In general terms, OAs improve or eliminate snoring in most cases and are an effective alternative treatment for OSA in a large number of patients, particularly those with mild-to-moderate OSA.[105]

The goal of therapy with OAs is to hold the upper airway structures in a position that enlarges or prevents collapse of the pharyngeal airway. There are two main types of OAs. Mandibular advancing devices attach to the teeth and force the mandible anteriorly, changing the dimensions of the upper airway. Radiographic studies of the awake airway have shown increases in the space between the soft palate and posterior pharyngeal wall,[106] the space between the base of the tongue and the posterior oropharynx,[107] or changes in the shape of the tongue and soft palate,[108] depending on the device examined. Many available devices are in this category, and the degree of advancement of the mandible varies considerably from device to device.

The second category of OAs are the tongue retainers, designed to hold the tongue anteriorly during sleep, preventing collapse back into the posterior airspace. The tongue is secured by suction into a plastic bulb located between the front teeth and lips.[109]

No trials comparing the effectiveness of the different OAs are available. A recent meta-analysis[105] of the various case series showed (1) all devices had similar success rates, with a 50% reduction in the number of apneas in 70% of patients, and a reduction to the normal range (defined as < 10 apneas or hypopneas/hour of sleep) in 51%, (2) oxygenation was improved to a similar degree, (3) success was related to the severity of the illness, with little success in patients with severe OSA or severe obesity (> 125% ideal body weight). Two recent studies comparing particular devices to CPAP in patients with mild-to-moderate OSA found CPAP to be more effective in treating the OSA but OAs to be better tolerated.[110,111]

Given the difficulties with compliance in using CPAP, OAs are a reasonable alternative for patients with mild-to-moderate OSA. A trial with an OA is indicated for patients with moderate-to-severe OSA who are intolerant of CPAP, are ineligible, or refuse other interventions. Because of the variability in individual response to OAs, it is necessary to document an adequate response in each patient with a sleep study.[112]

Intubation

The first treatment for OSA was tracheotomy, which bypassed the collapsed pharyngeal airspace. As an alternative prior to CPAP, a small group of patients was taught to pass a rubber nasopharyngeal (NP) tube coated with lidocaine gel into the pharynx, ending below the base of the tongue.[113] In another study of 24 patients,[114] only 44% tolerated the NP tube placement. Of those, two thirds had significant reductions in their OSA. Obese patients with severe apnea had the lowest rate of success. NP intubation is another option for patients with mild-to-moderate OSA who are unsuccessful with other forms of treatment, although patient acceptance is low.

Electrical Stimulation

Several investigators have explored the use of electrical stimulation of the pharyngeal dilator muscles to treat obstructive apneas. It is the sleep-onset-induced reduction in tone and work of these muscles that is the initiating pathophysiologic event in OSA. Electrical stimulation should lead to contraction of the muscles, dilation of the airway, and prevention of air-

way collapse. The results from current studies are conflicting. Two papers reported successful elimination of OSA and improvement in sleep continuity using topical electrodes to stimulate the genioglossus muscle[115] or needle electrodes near the hypoglossal nerve.[116] Apneas were terminated by application of stimulation after a preset interval of no airflow or prevented by intermittent stimulation. Other investigators have found that stimulation intensities required to terminate the apneas also caused arousal from sleep, suggesting that the arousal rather than increased muscle tone ended the apneic event.[117,118] Most recently, one group has achieved success in terminating or preventing apnea, without arousal from sleep, by utilizing deep fine needle electrodes placed into the genioglossus muscle or surgically placed electrodes around the hypoglossal nerves.[119,120] Electrical stimulation is a potential new therapy for OSA that requires further investigation to determine how widely applicable a modality it will be and which patients will benefit from using it.

Nasal Splints

Nasal splints were proposed as a method of dilating the anterior nares, improving nasal airflow, and decreasing snoring and, possibly, obstructive apneas. Subjective reports from patients using the Nozovent™, an internal nares splint, suggested a significant decrease in snoring.[121] However, objective measurement showed only a minor effect on snoring and no impact on obstructive apneas.[122]

Summary

There are many alternatives to the use of CPAP or surgery in the treatment of OSA. Weight loss can be curative in obese patients, the largest group of OSA patients, but is difficult to achieve and even more difficult to maintain. Positional therapy is effective for those patients with position-dependent OSA; however, position dependency should be demonstrated before initiating this form of treatment. Long-term outcome studies are not available for this form of treatment. All patients should be counseled to avoid factors that promote upper airway collapse such as alcohol, sedatives, narcotics, sleep deprivation, and cigarette smoking. Protriptyline and fluoxetine reduce the number of apneas in patients with mild OSA, but individual response is variable and should be validated. There is no role for any other agent as primary treatment for OSA, except to treat an underlying contributing condition such as hypothyroidism. Supplemental oxygen may ameliorate hypoxemia but does not relieve upper airway obstruction or improve symptoms and should be used only as an adjunct therapy. Oral appliances are an effective treatment for mild-to-moderate OSA and may have better patient acceptance than CPAP, but appropriate response to treatment should be documented. Electrical stimulation of pharyngeal muscles may prove to be an effective therapy but still requires further investigation.

Patients with mild-to-moderate OSA are the best candidates for the nonsurgical alternatives, whereas patients with severe OSA should have a trial of CPAP. Behavioral therapies can be initiated as primary therapy but should also be used in conjunction with other forms of treatment. OAs can be utilized as first-line treatment for mild-to-moderate OSA patients and may be useful in patients intolerant of other forms of therapy.

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Discussion

Brooks: How does tobacco make sleep apnea worse? I remember some studies that I think Kingman (Strohl) did that suggest nicotine as a treatment for sleep apnea.[1]

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Epstein: It's not clear what cigarette smoking does. One theory is that it causes mucosal edema; therefore, it narrows the upper airway. But, the actual mechanism is unknown. Maybe Kingman can answer this better. There was some thought that nicotine might be a respiratory stimulant or that it might increase respiratory muscle tone, but I think most of the studies have not shown a profound effect on apnea.

Strohl: The nicotine effect, because of its half-life, can be quite short.

Epstein: After about 1-2 hours you lose the beneficial effect.

Strohl: Nicotine patches have not been shown to produce either clinical or electrophysiologic improvement. The role of nasal inflammation in the pathogenesis of sleep apnea has a stream other than tobacco. Nasal lavage in sleep apnea patients shows higher degrees of nasal inflammatory cells, as do studies of tissues from surgery that show more inflammation and pathology. There are data showing that the nitric oxide concentration in gas exhaled through the nose is higher in sleep apnea patients, suggesting another inflammation hypothesis. There may be some role for the use of nasal steroids, either for enhancing compliance with nasal CPAP or even in mild-to-moderate snoring associated with cigarette smoking—anti-inflammatory approaches. The nicotine effect is not there from smoking, but the inflammatory effect is in the airway.

Woodson: I've had a moderate experience with oral appliances, and they can work quite well. The most successful devices tend to be adjustable. To get a therapeutic response to an oral appliance, you usually want to get the lower jaw moved out anteriorly enough to have at least end-to-end approximation of the incisor teeth. A good initial aim of therapy is about 75% of maximal protrusion. However, in many apneics, you won't see a response, and you need to go farther. But if you rapidly advance too far, you'll start having problems with the tempromandibular joint. So, with an adjustable device you slowly advance and improve the patient's tolerance to the device. It's also important to have a device that allows people to brux. If you take any joint of the body and freeze it in a single position all night, it will be pretty sore the following morning. Being able to actually move the joint a little bit dur-

ing the course of the night is very important. So, a lot of the more effective devices don't try to lock someone into a single position, but allow some mobility of the lower jaw.

Epstein: I agree. The more you can pull out the jaw, the greater the effect on the airway and the more likely you are to get improvement in apnea. There are several approaches people are taking. A number of devices allow one to sequentially increase the pull on the mandible. One device actually has a servo motor that can be activated during a sleep study to determine what degree of advancement gives the maximal effect. Others have a range of positions that can be set and slowly increased over time. Another approach is to use a combination of devices. Initial treatment is started with an appliance that gives a small amount of protrusion, like the snore guard, and as people adjust to that, they move on to an appliance that gives greater anterior positioning of the mandible.

Brooks: Do the oral appliances affect sleep?

Epstein: The oral appliances can improve or impair sleep architecture, depending on how well people adjust to them. They can cause significant pain and discomfort, leading to fragmentation of sleep. As with CPAP, as one begins to adjust to the device, there is less fragmentation of sleep. The oral appliances also can improve sleep. As apnea is reduced, sleep fragmentation is reduced and sleep architecture is improved.

Brooks: I wonder if the mechanism would be similar to the tricyclics. You breathe better because you're not sleeping as deeply.

Epstein: Not that I'm aware of.

Stoller: I have a couple of practical, clinical questions. Number one, Do you order thyroid function tests on every patient in whom sleep apnea is suspected? Number two, If you've made an intervention, such as the ones you described or conventional CPAP or even surgical therapy as we will hear, how do you decide when to reassess the patient? How much weight loss should the patient undergo before performing a follow-up study? Should that follow-up study be a full center-based study or do you use surrogate measures of subjective wakefulness to decide? How do you manage the longitudinal aspects in these patients and would you check the thyroid functions up front?

Epstein: I used to check everybody's thyroid function. There are recent data from Winkelman et al at Brigham concerning this practice.[1] They found that unless the history and physical exam suggested the presence of hypothyroidism, screening with routine thyroid function tests was not helpful. Their recommendation is that you should think about the possibility of concurrent hypothyroidism in any overweight patient with sleep apnea. However, if you don't find anything on history and physical exam suggestive of hypothyroidism, then you probably aren't going to pick up with routine blood tests more patients who have surreptitious hypothyroidism. In terms of weight loss, it's hard to predict in advance how much weight any individual patient is going to need to lose to eliminate sleep apnea. Usually we'll select a target goal related to an individualized weight-loss program. When they reach their target, that's when I'll tell them, "Stop wearing your CPAP for a little while and see how you feel." If they maintain their improvement in symptoms, I bring them back to the sleep lab to see: (1) if the apnea is still present; or (2) if it is present, is the severity reduced sufficiently for them to be eligible for one of the alternative therapies if they don't like CPAP, such as a dental appliance. Additionally, less severe disease may allow a reduction of CPAP pressure, hopefully improving comfort and promoting continued use of their CPAP.

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Chediak: I have two questions. The first one is Do mandibular repositioners improve sleep apnea by increasing airway size, tensing the upper airway tissues, or changing the length/tension relationship of the upper airway dilators? The second question is In the patient with very severe OSA, in the absence of important physiologic consequences such as heart disease, and in whom the primary complaint is "I'm sleepy," what's wrong with drugs that may keep him awake, such as modafinil?

Epstein: With regard to the mechanism of action of the oral appliances, the studies that I'm aware of show that the devices change the size of the upper pharyngeal air space. Alan Lowe et al[1] used 3-dimensional reconstruction of CT scans to show that their device increases mostly the anteroposterior dimensions of the upper airway. Dr Erman, are you aware of any other studies looking at things such as muscle tension relationships? I'm not. The only studies that I know of looked at changes in airway diameter. With regard to treating patients who are asymptomatic with stimulants, one would question why, if they are asymptomatic and have no hemodynamic consequences, are you treating them?

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Chediak: Well, I didn't say asymptomatic. I said sleepy patients who do not have serious physiologic cardiovascu-

lar or pulmonary consequences. One would like to treat excessive daytime sleepiness. This is a strategy that I have used in patients with periodic limb movements in sleep and sleep fragmentation with excessive daytime sleepiness in whom other conventional pharmacologic agents for that disorder have failed.

Epstein: This brings up the philosophical question of whether to treat a symptom or the underlying disease that is causing the symptoms. We all know that we run into patients who, regardless of what you do, will not accept therapy. At that point you're left with a choice. Do you treat their sleepiness or not? I think we're going to see more of that because medications such as modafinil, reputed to have fewer side effects and abuse potential, are now available. It is possible to take your patients with mild apnea and just give them a stimulant to keep them awake. My approach would be to try to treat the underlying disorder, but I'm not sure that I can give you an evidence-based outcome or cost-effectiveness rationale for doing that. How do the rest of you approach those folks? Anyone else want to tackle that?

Strollo: I have treated people with stimulants in the past but mostly not as an alternative to positive pressure, but in people with residual daytime sleepiness. There are, clearly, a subset of people who are objectively compliant with positive pressure and still remain sleepy. That's their primary complaint. I'm fairly rigid about how I approach these folks. I usually do an MSLT (multiple sleep latency test) and document objective daytime sleepiness and objective compliance with positive pressure. In those folks I haven't used modafinil because it hasn't been available to me, but I have used ritalin and occasionally dexedrine. I know other people who do that. John Shepard does that. You may do it, Alex (Chediak), as well. When the complaint is disabling sleepiness, I think that's an approach that one has to consider.

Epstein: We all know from dealing with these patients that when they come in and say "I'm using the CPAP, but I'm still sleepy," Reasons 1-10 for why they are still sleepy are noncompliance with their treatment. If they are compliant, the next most common reason is insufficient amounts of sleep. I think your approach is appropriate—to confirm the patient is compliant with therapy and is getting appropriate amounts of sleep. Objectively document continued sleepiness before giving them medications, which do have side effects. Stimulant medications increase the risk of hypertension and have CNS [central nervous system] effects. I think you want to make sure that there's not something else you can do first.

Woodson: A large percentage of people who have sleep-disordered breathing are on the milder end of the spectrum. They're not the people who are at the severe end of the spectrum. So, frequently you have the clinical situation of having a person who is primarily a snorer who has very mild, if any, symptoms of sleepiness. You're going to consider treating them with an oral appliance. Do you need to get a sleep study? Or what kind of study and how do you approach them?

Epstein: The question of how to diagnose is the same one we debated yesterday. There is really no difference in the approach whether you are considering an oral appliance or CPAP or surgery as treatment for suspected sleep apnea. The reason to get a sleep study is to document both occurrence of disease and severity. If you're considering an oral appliance, it's still important to document that they are in the range that is appropriate for treatment with an oral appliance. An extra level of complexity is added by the need to document an appropriate response to therapy with the oral appliance. If anything, I think you may end up doing another sleep study to prove that it worked. An asymptomatic snorer does not require a sleep study before initiating treatment with an oral appliance or any of the other available therapies.

Woodson: Actually, if you have a patient coming in really complaining of snoring who has no symptoms of sleepiness, the problem, from a practical point of view, is that frequently the third-party payers will refuse to allow a sleep study, which is a problem.

Strollo: Current indications for polysomnography address that, and I believe the recommendation is that only symptomatic snorers should get a sleep study. For an asymptomatic snorer, polysomnography is not recommended. That gives you a little wiggle room in terms of ...

Strohl: That comes from population-based studies in which there are many people who by most criteria, short of doing MRIs [magnetic resonance imaging] and cardiac echoes, are healthy and have either heavy snoring or high apnea-hypopnea indexes. Now they may be in some early stage of their illness, but heavy snoring by itself, without other symptoms, is not uncommon. Most people who present to a physician have a complaint. But going out and saying "Do you snore?" is an advertisement to get a lot of people who don't have much of anything other than a loud, noxious noise. It has to be thought of very carefully, for these people will take up health care resources to a greater extent than those with car accidents, angina, or uncontrolled hypertension from sleep apnea, a more limited population currently unrecognized.

Epstein: In this patient population you can treat the symptom. If you're only treating snoring, you don't need to bring them back for a sleep study with the appliance to document that it works. You just need to ask the patient or bed partner "Is the snoring gone?"

Strollo: The other practical aspect, though, is that in most health care systems and insurance arrangements, if you

don't have sleep apnea, medical insurance will not pay for an oral appliance. So, the patient has to bear the cost of that. I just wanted to share with you just a little bit of data that was presented in Edinburgh just a couple of weeks ago. Alan Lowe and the group from Canada are putting together a study to look at a comparison between CPAP and oral appliances, long term. They're trying to determine if there is a difference. They randomize the patients to CPAP and oral appliances, and look at compliance objectively. They have tried to do this with a microprocessor chip in the oral appliance that senses temperature change and therefore, you know whether it's in the patient's mouth and for how long. When this was first presented it sounded like a great idea. Apparently, they're on the fourth generation of the chip. It's very difficult to get the compliance meter to work properly. The other thing that's happened with the study and the data that were presented by John Fleetham is that the mean time to optimal treatment in those treated with oral appliances was 237 days, compared to 37 days from the first sleep study to optimal CPAP treatment. So, there are some factors to be considered. That may be the best-case scenario. The other thing is that there is a significant cost, at least in the way most oral appliances are provided in our community. In Pittsburgh, it costs approximately $1,000 to have an oral appliance fitted properly and adjusted during serial dental visits by a knowledgeable dentist. That approaches the cost of CPAP, so the patient needs to be aware of that. The health care system needs to be aware of that.

Brooks: For the patients you spoke of earlier who seem to be sleepy despite good treatment, there may be a place for something like an Actigraph to really demonstrate that they're getting enough nocturnal sleep. But I also have a question on fluoxetine. My understanding is that it does not work by suppressing REM, [rapid eye movement] but through other mechanisms. Is that true, and if so, how does that affect sleep apnea?

Epstein: There's really only one study that looked at the effects of fluoxetine, by Hanzel et al,[1] and they really didn't go into mechanisms. They reported reduction in REM sleep with fluoxetine but no difference from baseline in number of arousals from sleep. Other than that, the mechanism really hasn't been elucidated very well. There are some theories that serotonin, which is a sleep-inducing agent, may have some effects as a ventilatory and upper airway inspiratory muscle stimulant, but whether that's how fluoxetine functions in reducing the number of apneas isn't well studied.

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Brooks: Given its good safety profile, I'm surprised that it hasn't been used more. I'd be interested to hear why the people in this group don't use it, or if they do.

Epstein: The apnea-reducing effect of fluoxetine is just as mild as that of protriptyline, so I think the role for the antidepressants is rather limited. The response to these agents is quite variable, and the response of any individual cannot be predicted. So, despite the improved side effect profile of fluoxetine, this agent's role is in patients with mild disease who do not improve with other forms of therapy.

Erman: In addition to protriptyline, other tricyclics can work. Nortriptyline, which is less anticholinergic and therefore, often better tolerated, is not as stimulating as protriptyline, but can work and is helpful for some patients. The problem is that the effects are rather mild. It's not an effective treatment for the vast majority of patients with significant apnea. There are also problems with patients tolerating treatment, even with the fluoxetine, which may also produce effects of lightened and disrupted sleep. Overall, the effects are not terribly strong, so medications are usually only appropriate for rather mildly affected patients.

Phillips: In the space of a week I had a television reporter, a patient, and the chair of my department call me and ask me about somnoplasty. You look perplexed, as I was. I said quite frankly "I've never heard of this." If the people in this room don't have much experience or expertise in this, I think maybe we need to be up front with it. Does anybody here want to talk about somnoplasty?

Epstein: I think Dr Woodson is going to talk about that in his discussion of surgical treatments.

Strohl: The fluoxetine story is that there was a variable response.[1] Hanzel did not push the dose to the levels used to treat depression because these patients did not meet clinical criteria for depression, but the thought is that you could modulate specific neurotransmitters at the hypoglossal nerve motor unit and selectively or at least preferentially activate upper airway muscles and still have the person sleep. Pharmacologic therapy may come as a consequence of our greater knowledge of the neurobiology of how that unit is to keep the upper airway in an awake state and let the brain sleep. But that's tough to accomplish right now because of the interrelationships between these systems.

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Alternatives to Positive Pressure: Surgical Therapy

B Tucker Woodson MD FACS

Introduction
Upper Airway Structure
Airway Evaluation
Criteria of Success
Surgical Treatment Approaches
Specific Surgical Procedures
Perioperative Care
Summary

Introduction

A role exists for effective surgical treatment of obstructive sleep apnea syndrome (OSAS). A variety of medical treatments have been described including weight loss, positional therapy, oral appliances, and nasal continuous positive airway pressure (CPAP). It is accepted that nasal CPAP is the primary mode of therapy. However, nasal CPAP and other medical treatments are not uniformly successful. Patients may fail, poorly accept, or poorly tolerate medical therapies.[1,2] Although failure rates may be ameliorated and reduced by aggressive intervention, they frequently fail to respond.[3,4] For those patients failing medical therapy and for those patients with overt upper airway (UA) pathology, surgical treatment is an appropriate alternative. Improvements in surgical technique and selection methods may offer more individuals surgery as a potential primary therapeutic alternative.

It has been demonstrated that for most patients, OSAS is associated with structural abnormalities of the UA. OSAS results from the combination of a structurally small UA and the loss of muscle tone related to sleep. The UA is smaller and more collapsible in those who experience sleep apnea compared to those who do not.[5-9] Increased velopharyngeal collapsibility has been correlated to the apnea-hypopnea index.[10] The closing pressure (defined as the UA pressure at which airway cross-sectional area or inspiratory airflow approach zero) is a measure of collapsibility. Closing pressures controlled for the level of muscle tone are higher (ie, more positive) in those who experience apnea than in those who do not.[11] For structural abnormalities, it seems logical to propose an anatomic solution, such as surgery. The size of the UA is determined by the dilating forces of the airway interacting with the collapsing forces.[12] Surgery alters the balance of the collapsing and dilating forces.

Upper Airway Structure

UA obstruction may be described partially by structural changes that occur during development. The human UA is unique compared to that of other mammals. It differs in having a long, collapsible pharyngeal segment. In other mammals and in normal human infants, the cartilaginous larynx (which provides a solid UA framework) is located high in the neck. This cephalic position is close to the skull base and produces a highly stable airway. Even with loss of muscle tone and increased negative inspiratory forces, a cephalic UA position is resistant to collapse and obstruction. With cranial and facial development, the laryngeal position changes.[12] Skull flexion increases with cranial enlargement. With increasing skull flexion, the laryngohyoid complex descends more inferiorly into the neck.[13] The final result is a larynx that is separated from the base of the skull. Whereas in infants, the

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larynx is located approximately at the level of the body of the second cervical vertebra, in adults it is located approximately at the position of the body of the fifth cervical vertebra. Moreover, in subjects with OSAS compared to normal subjects, the laryngohyoid complex is even more inferiorly displaced to the level of the body of the seventh cervical vertebra.[14] A longer soft-tissue supralaryngeal airway results, which requires increased muscular activity to help maintain patency. In OSAS, the smaller diameter and longer length contribute to airway instability.

The cross-sectional area of the UA is smaller in OSAS compared to normal subjects.[7,8] Constricted segments vary but may involve the nasopharynx, oropharynx, and hypopharynx.[9] Early studies that attempted to evaluate UA size were confounded by differences in patient populations and fluctuating UA muscle tone. Studies controlling for UA muscle tone and with age- and weight-matched controls have demonstrated structural differences between apneic and nonapneic populations.[5,15]

The most vulnerable segment due to size and collapsibility is the velopharynx in the area behind the soft palate.[7] This retropalatal airway is the smallest and most collapsible segment both in apneic and nonapneic individuals.

Although anatomically small, the UA in adult OSAS generally does not exhibit overt pathology.[16] This is in contrast to children in whom tonsillar and adenoidal hypertrophy and other lymphoid hyperplasia frequently are the etiology of OSAS. Treating this pathology especially in children may be successful. However, in most adults, abnormal airway characteristics are more diffuse. Subtle differences in skeletal and soft tissue structure exist. Cephalometric x-rays demonstrate skeletal abnormalities including maxillary retrusion, mandibular retrusion, and increased mandibular plane angle.[14] Other findings include incisor overjet and increased anterior facial height. Not all of these findings are necessarily present in a single individual but are more descriptive of populations of patients with sleep apnea or snoring. The sum of observations describes a population with an elongated pattern of facial growth. Facial growth patterns are elongated more inferiorly rather than anteriorly. A relative maxillary retrusion results in a smaller posterior airway.

Soft tissue findings include a long and wide soft palate, a large tongue, a low position of the hyoid, and a narrowed respiratory disturbance index (RDI).[18]

Endoscopy, manometry, magnetic resonance imaging studies, and videofluoroscopy demonstrate patterns of UA collapse that are complex. The primary location of obstruction is the retropalatal airway; yet, it is infrequently the only location of obstruction. In only 20% of patients is the retropalatal airway the isolated area of collapse.[19] The palate is the primary location of obstruction in 20-45% of patients. In 10%, obstruction is only at the tongue base. In many patients (45-55%), obstruction involves multiple airway segments. Collapse often is initiated in one segment and progresses inferiorly in the airway as negative inspiratory pressure is developed.[20,21] The pattern and the level of obstruction vary between patients and even in the same patient, depending on body position and sleep stage. A classification of airway types and areas of obstruction is provided in Figure 1.

The pattern of collapse within airway segments is complex and only incompletely understood. Collapse depends on airway shape and size and on the properties of the surrounding tissues.[22] It may be influenced by the properties of the lateral pharyngeal wall.[23] Surgical procedures that are more effective in changing the shape of the airway and influencing collapse of the lateral wall may be more effective.

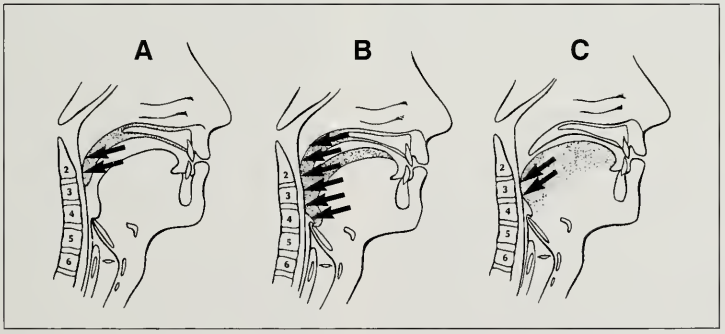

Fig. 1. Common patterns of obstruction: **A.** Isolated palatal collapse (Type I) occurs in 20-40% of patients. **B.** Combined palatal and tongue obstruction (Type II) occurs in approximately 40% of patients. Collapse of the tongue and palate may occur simultaneously or sequentially. The lower pharynx may close secondarily as negative inspiratory pressure collapses the airway. **C.** Primary lower pharyngeal obstruction (Type III) occurs in approximately 10% of patients. From the American Academy of Otolaryngology, with permission.

Airway Evaluation

Many methods to evaluate the UA and guide surgical treatment have been described. Most methods are imprecise and fail to account for the dynamic nature of the pharyngeal conduit. No methods of evaluation have been demonstrated to be useful positive predictors of surgical outcomes. Clinical evaluation methods include pharyngeal manometry during sleep, pharyngeal endoscopy (both in the awake and sleep state), and physical examination. Other methods are described but have not guided surgical therapy. There is no consensus as to which, if any, method is best.

The level of airway collapse as identified on clinical evaluation does predict surgical outcomes. Sher et al[24] used a meta-analytic technique to assess the literature on uvulopalatopharyngoplasty (UPPP). The site of obstruction did predict outcome. In those patients with tongue-base obstruction, surgery was successful only in approximately 5%. This was in contrast to patients with palatal collapse who were not considered to have tongue-base collapse in whom surgical success was 40-50%. At least one study suggests that the response to Müller's maneuver can be predictive.[25] Müller's maneuver, a forced inspiratory effort with the mouth and nostrils occluded, is performed during endoscopy. Percent collapse is then estimated. The presence of tongue obstruction predicts failure; however, the absence of tongue obstruction does not predict success. Other clinical features predicting outcome include apnea severity and body mass index. Patients with severe apnea (an RDI > 40 events/h) may have poorer long-term success rates.[26] Very obese patients have a higher risk of UPPP failure.

There are many reasons for the poor predictive value of airway examination. The site of obstruction during sleep (determined by airway manometry and endoscopy) correlates poorly with endoscopic Müller's maneuver of the pharynx.[27,28] Cephalometric x-rays, which are a more quantitative measure of the airway, have not been compared to the site of UA obstruction during sleep. With better knowledge of the UA, it is hoped that evaluation methods will improve.

Criteria of Success

There is a large experience with UPPP for the treatment of sleep-disordered breathing. The procedure reduces sleepiness and snoring in a large number of patients. Objective respiratory measures demonstrate poorer results; however, the subjective outcomes of UPPP should not be entirely dismissed. It is accepted that apnea and hypopnea indices, low oxyhemoglobin desaturation, and sleep fragmentation correlate only poorly with sleepiness.[29] A discrepancy in outcome measures following surgery is not surprising. Objective respiratory measures are the most important outcome measure in OSAS, but others may also be valid. Total elimination of respiratory disease may not be required to decrease sleepiness. Zorick et al[30] objectively measured sleepiness using the multiple sleep latency test, or MSLT (with response defined as a 50% decrease in RDI). Patients in whom nasal CPAP had been documented as effective subsequently underwent UPPP. Both treatments produced equivalent improvement in MSLT scores. This compared to UPPP patients with < 50% decrease in RDI (ie, non-responders) who demonstrated no change in MSLT scores. The use of RDI as a measure of apneic severity is also confounded by variables such as age and fluctuating definitions of hypopnea and other nonapneic respiratory events. For example, an RDI of 5 in a child of 5 years of age (which is likely pathologic) cannot be compared to a similar index in an asymptomatic 60-year-old subject in whom the same RDI may be considered trivial.

Few data correlate cardiovascular morbidity or mortality with apnea severity. He et al[31] associated a higher mortality rate with an apnea index of > 20 events/h. Because hypopnea indices were not included in this study, comparisons to RDI are speculative. RDI mortality thresholds are likely higher. Lavie et al[32] were unable to associate any level of RDI with increased cardiovascular mortality. The effect of UPPP on mortality is controversial. Keenan et al[33] were unable to identify a difference between nasal CPAP mortality rates and UPPP mortality rates.

Just as there are no criteria to determine a threshold for adequate duration of nasal CPAP use; no criterion to determine a threshold for adequate surgical outcome exists. No single measure discriminates normal patients from those with mild, moderate, or severe sleep apnea. In general, describing a condition as normal-to-mild would be expected to be associated with minimal symptoms and no or very low levels of significant medical morbidity. At the other extreme, severe disease suggests marked symptoms and a demonstrable risk of medical morbidity and mortality. Moderate disease severity is of an intermediate nature. Because of the problems in determining thresholds of disease, the criteria for surgical success are arbitrary. They will undoubtedly evolve. Possible definitions of OSAS severity may include (but are not limited to): patient subjective symptoms (excessive daytime sleepiness, snoring), objective sleepiness (MSLT, MWT [maintenance of wakefulness test]), objective respiratory measures (apnea index, hypopnea index, oxyhemoglobin desaturation), sleep stage measures (non-rapid-eye-movement sleep, or non-REM vs REM sleep), cardiovascular morbidity, and mortality. Impaired driving performance is a critical outcome. Data suggest that both nasal CPAP and UPPP decrease driving risk.[34] For UPPP, lowering the driving risk has been demonstrated using advanced driving simulators, vigilance tests, and studies of population accident rates.[35,36]

A consensus on threshold may not exist, but clinicians need criteria of disease severity for therapeutic guidance. Some surgeons have used treatment thresholds for nasal CPAP as a definition to guide adequate surgical treatment. Patients without symptoms and an RDI < 15-20 events/h frequently are not treated with nasal CPAP. This level does not imply a cure

but rather a treatment threshold. The comprehensive review of Sher et al[24] used similar criteria (a > 50% decrement in respiratory events plus either an RDI of < 20 events/h or an apnea index of < 10 events/h). As definitions of disease change so will definitions for adequate treatment.

Surgical Treatment Approach

Surgery is postulated to be effective by modifying the volume, shape, compliance, or collapsibility of the UA.[37,38] When surgery effectively modifies UA critical closing pressure, it successfully reduces RDI. The surgical approach to achieve effective changes differs according to the severity of disease, symptoms of the patient, and philosophy of the surgeon. In general, surgical objectives include (1) removing or altering overt pathology (ie, tonsils, adenoids, cysts, or tumors); (2) selecting patients with discrete sites of airway obstruction, which are then modified; and (3) treating the global airway irrespective of differing sites of obstruction.

Overt pathology is often present in children, and OSAS commonly improves following its removal. Results in adults are much more variable. Rarely are tonsillectomy and septoplasty alone successful when performed as isolated procedures. The exception is when they address an isolated abnormality in a patient with mild or moderate disease.[39] Follow-up polysomnography is required postoperatively to document effectiveness.

The second approach attempts to identify specific segments of airway obstruction. In this site-directed approach, critical areas of collapse and obstruction are identified. This is not uniformly successful. Most studies have been unable to positively predict success. The exception is the methodology of Launois et al,[40] which controlled for muscle tone and physiologic variability of the UA. Once critical areas are identified, these are modified singularly, in combination, or in a staged sequential fashion, depending on the needs and desires of the patient and the surgeon. An example would be a patient who is considered to have primarily oropharyngeal obstruction without involvement of other sites. In such a patient, an isolated UPPP may be a consideration. Polysomnography is then repeated, and if disease is persistent, a residual obstruction can be treated with other techniques.

In an approach that is not site-directed, evaluation of the UA is not considered as critical. The entire UA is addressed in the initial procedure. This may involve combined pharyngeal procedures or bimaxillary advancement. In practice, all three are often used. The first approach is used in patients with overt pathology. The second is used in mild-to-moderate OSAS or snorers with clearly defined narrowing. The third approach is used in surgical failures, severe disease, or the presence of multiple risk factors contributing to airway collapse, such as morbid obesity. The ideal approach maximizes success while minimizing overtreatment, cost, and surgical morbidity. The concept of describing the effects of surgery (or other intervention) using critical closing pressures has been described.[11] In this model, the airway is viewed as a collapsible tube. Patients without apnea have more negative critical closing pressures while patients with severe apnea have positive UA closing pressures. Snorers and patients with upper airway resistance syndrome are in between. Effective airway interventions decrease closing pressures from the apneic into the normal range. Less effective interventions do not produce adequate change in critical closing pressures. It is speculated that multiple procedures can be combined to produce an additive effect on the UA for the desired outcome (Fig. 2).

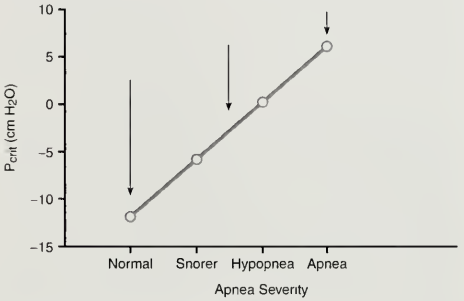

Fig. 2. Critical closing pressures (P_{crit}) and apnea severity. P_{crit} is the level of nasal continuous positive airway perssure (CPAP) at which maximal inspiratory airflow becomes zero. In practice, P_{crit} is the level of nasal CPAP that initiates opening of the airway during a CPAP titration. Effective interventions shift P_{crit} to the left (more negative). An individual with apnea may start at a positive P_{crit} (small arrow). A limited surgery or weight loss may partially improve the airway (medium arrow). Success is achieved when P_{crit} approaches the large arrow.

Because snoring is a noise produced by vibration of the palate and lateral pharyngeal tissues, surgery for snoring may have an effect different from apnea surgery.[41,42] Snoring may be reduced by excising vibratory tissues or increasing palatal stiffness. This shifts the low frequency formats associated with snoring to a higher frequency.[43] Alternatively, removing areas of obstruction may reduce negative inspiratory forces and eliminate flutter and vibration.

Specific Surgical Procedures

Uvulopalatopharyngoplasty

UPPP has, almost since its inception, been controversial. The procedure involves excision of the uvula, a variable portion of the distal soft palate, the faucial tonsils, and a portion of the tonsillar pillars and pharyngeal mucosa.[44] Success with UPPP varies according to outcomes measured. For snoring, the procedure has a reported success of 80 to 90%. Variable rates of relapse have been reported.[45] It is commonly accepted that, at best, one half of patients following UPPP have ade-

quate clinical reduction in respiratory disturbance. The meta-analysis by Sher et al[24] analyzed 9 papers containing raw preoperative and postoperative data (168 patients) that also presented information on location of collapse. In this group, UPPP success was 40.7% (using the criterion of an apnea index of < 10 or an RDI of fewer than 20 events/h combined with a > 50% decrease in apnea or hypopnea). UPPP success was correlated with both severity of disease and location of collapse. Successful responders had significantly lower severe apnea indices (31.2 ± 23.3 events/h vs 56.6 ± 30.5 events/h) and RDIs (43.1 ± 26.3 events/h vs 65.7 ± 26.7 events/h). Location of collapse affects results. Patients with upper pharyngeal obstruction (Type I) demonstrated a 52.3% response rate and a 32% reduction in RDI. This is compared to those with lower pharyngeal obstruction (Type II) who demonstrate only a 5.3% response rate and a 6.5% decrease in RDI

Long-term response rates following UPPP vary. Larsson et al[46] reported 6-month success rates of 60%. This decreased to approximately 50% at a mean of 46 months postoperatively. A higher rate of relapse occurred during an interim 2-year study and was associated with an increase in body mass index. Janson et al[26] noted similar respiratory results with 64% initial success and 48% long-term success at > 4 years. In this study, no change related to body mass index was identified. Of note, no patient with an RDI > 40 events/h demonstrated long-term success.

Using alternative outcomes, UPPP has been associated with a reduction in α-electroencephalographic (EEG) arousals in nonapneic snorers.[47] It has also been associated with improvements in cardiac ventricular function.[48] Concerns have been raised that UPPP may increase problems with mouth leak on subsequent nasal CPAP attempts.[49]

To improve success rates, attempts have been made to identify only those patients with palatal obstruction. Patients who are likely to fail (those with obstruction at other pharyngeal sites) are excluded. Most patient selection methods identify retrolingual obstruction, either directly or indirectly. Cephalometric x-rays have been advocated, and many differences between responder and nonresponder groups have been retrospectively identified. None have been prospectively predictive.[50-53] A prospective evaluation by Dograhami et al[54] using fiberoptic endoscopy and cephalometrics failed to identify significant differences between UPPP responders and nonresponders. Palatal-length measures approached significance. Studies suggest that stratification by skeletal subtype may be important when using skeletal measures.[55] Also airway length may be an important characteristic instead or in addition to anterior-posterior width.[56] Other techniques such as UA manometry have been used but with little positive predictive success.

Uvulopalatoplasty

Uvulopalatoplasty (UPP) is a procedure directed at the palate and the uvula that was initially introduced for the treatment of snoring but has now been extended for the treatment of sleep apnea. The procedure has many technical variations and may be extended to remove pharyngeal folds and tonsils. In selected patients, UPP may be similar to traditional uvulopalatopharyngoplasty, or UPPP. Other modifications include midline palatal mucosal stripping with subsequent scar contraction, which shortens and stiffens the palate. The techniques may be performed with laser, electric cautery, or traditional surgical techniques. My colleagues and I initially described the laser procedure (LAUP).[56] The advantage of the laser is that it allows simultaneous cutting and coagulation. These characteristics are not unique to the laser, and other technologies may be used. The procedure (Fig. 3) can be performed in the ambulatory care setting of the physician's office under strictly local anesthesia—a major advantage.

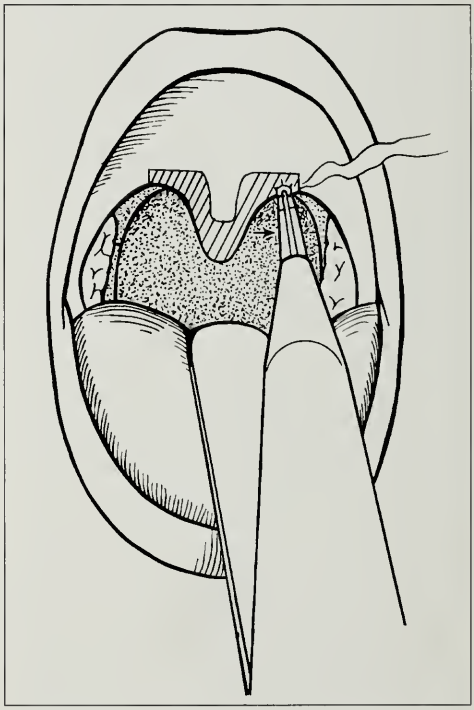

Fig. 3. Uvulopalatoplasty with laser. A portion of the palate and uvula are removed. Usually the procedure is repeated over several sessions. From the American Academy of Otolaryngology, with permission.

Studies have reported snoring outcomes with approximately 80% short-term success rates.[57,58] These reports are based on subjective complaints of bed partners and are not actually

based on noise reduction. Evaluation of snoring noise before and after surgery indicates that there is a shift in the spectrum of the noise towards high frequency, fricative sounds in many patients. Only in a small percentage is the snoring noise eliminated. Because it is the complaint of the spouse that is crucial regarding snoring, for most patients the effect on spousal complaints may be the most clinically important outcome.

Complication rates for a UPP are low.[59] The most common complaint is that of a sensation of increased mucus or phlegm. Longstanding speech or swallowing complications are unusual. The major morbidity with the procedure is perioperative pain, which may be severe. The long-term effects are also unknown. It is also possible, depending on the technique that is used, to create a partial pharyngeal stenosis.[60] It is therefore conceivable that sleep apnea could be worsened.

As with traditional UPPP, there is controversy about the effectiveness of UPP for OSAS. Because for many patients the procedures are technically similar, it could be speculated that the results would also be similar. In general, studies of UPP patients have included patients who are less obese and with milder disease than those studies using traditional UPPP.[61] Because severity of disease is a critical predictor of outcome, it is difficult to compare the techniques. Studies have not consistently shown an effect on RDI, but a small subset of patients (approximately 40%) have a significant reduction of RDI to clinically acceptable levels.[59,61,62] LAUP has also been associated with statistically significant improvements in the duration of respiratory events, microarousal index, and duration of oxyhemoglobin desaturation.[63,64] Initially, there was concern that there was potential for significant worsening of apnea following LAUP. Objective evaluation of airway size has not demonstrated significant acute airway obstruction following the procedure.[65] Respiratory disturbance may acutely worsen; therefore, when it is applied in patients with OSAS, perioperative CPAP is recommended.

Surgery of the Lower Pharynx

There are 2 general surgical approaches to address the lower pharynx. First, the skeletal and facial UA structures may be advanced and modified. This enlarges the airway by moving pharyngeal muscles and muscle insertions. Second, soft tissues may be excised, such as by glossectomy. Although it has been postulated that these procedures have the effect on the lower pharynx, the most important effect may be indirectly on the velopharynx.[66]

Maxillofacial Surgery

Maxillofacial surgery described for OSAS includes: (1) segmental osteotomies, which move portions of the mandible that are not teeth bearing, (2) mandibular advancement with movement of the entire teeth-bearing mandible, and (3) bimaxillary advancement, which advances both the maxilla and the mandible.

The anterior wall of the pharynx is composed of the tongue and related structures. The position of the tongue is determined partly by the position of the genioglossus and geniohyoid muscles and their insertion onto the genial tubercle. Traditional mandibular advancements for OSAS include the teeth-bearing segments, which may restrict the amount of movement attainable unless specialized osteotomies or aggressive orthodontia are included. Such procedures are limited by dentition or require major orthodontia. To avoid this, limited mandibular osteotomies were developed that advanced only the insertion of the genioglossus or geniohyoid muscles (Fig. 4). Many different osteotomies have been described to accomplish this.[67,68] The genioglossus muscle insertion is superiorly located on the genial tubercle, and osteotomies need to be superiorly located near the alveolus. Aggressive genioplasties using traditional techniques may risk missing the insertion point or creating an unstable mandible with a possibility of mandibular fracture. Modified techniques preserve the inferior mandibular border and minimize the risk of fractures. Hyoid advancement procedures may modify the position of the tongue base and tense the lateral pharyngeal walls. The hyoid may be advanced to the inferior border of the mandible or alternatively pulled anteriorly and inferiorly to the larynx or thyroid cartilage. Only a single study[69] has evaluated hyoid myotomy performed as an isolated procedure.

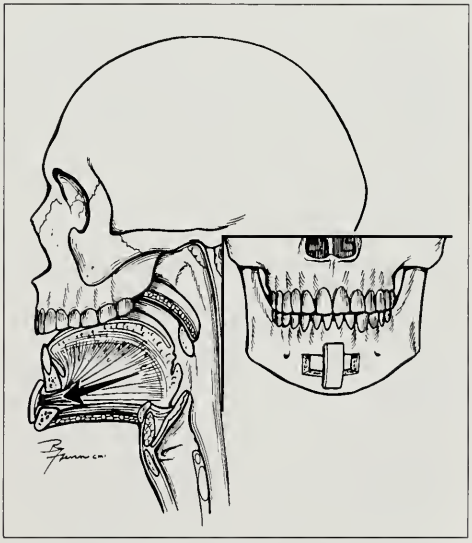

Fig.4. Limited mandibular osteotomy. The genioglossus insertion is advanced anteriorly without movement of the mandibular dentition. From the American Academy of Otolaryngology, with permission.

Multilevel Surgical Results

Several studies now support that multilevel surgeries have improved results compared to UPPP alone. UPPP results in patients with lower pharyngeal obstruction (Type II) have success rates from 5-10%. With the addition of limited skeletal procedures to UPPP and Type II UA, success has been reported to be from 60-67%.[63,70,71] Studies using only genioglossus advancement without hyoid suspension have reported similar results.[71] Recent studies have demonstrated an approximate 60% success rate (defined as an RDI < 20 events/h and a > 50% reduction) and have also demonstrated improvement in other outcome measures such as the Epworth sleepiness score.[63] Hyoid myotomy performed as a separate procedure from UPPP or genioglossus advancement has also been described and demonstrated success in some patients.[69] It should not be advocated as an isolated procedure because it has been performed in patients who have previously had other surgical procedures such as UPPP. A randomized study that included controls, nasal CPAP, and a surgical treatment arm demonstrated that RDI (measured as an oxygen desaturation index) was not reduced as significantly as with nasal CPAP. However, symptoms were reduced similarly.[72] This was in spite of more severe disease in subjects in the surgical arm. As with UPPP, patients with morbid obesity do less well with multilevel surgeries.

Bimaxillary Advancement

Several studies have demonstrated the effectiveness of bimaxillary (maxillomandibular) advancement for OSAS.[70,73,74] These skeletal advancement procedures enlarge the airway by movement of both the mandible and the maxilla. In contrast to traditional orthognathic surgery, skeletal advancement procedures require a more aggressive approach. In retrognathic patients who are not obese, advancement of approximately 8-10 mm is associated with a high success rate, even without other soft tissue surgeries.[73] However, in patients who are more obese and who have other risk factors, success rates may be less. In these latter high-risk patients, bimaxillary surgery, combined with other techniques described previously, results in high success rates.[70] At the present time (1998), bimaxillary advancement is indicated as primary treatment in patients who have clinically important maxillomandibular deficiencies and who are not obese. In patients with morbid obesity and OSAS, maxillomandibular advancement is utilized as part of a staged protocol with greater than 90% success. Complications of bimaxillary surgery include change in appearance, facial parasthesias, change in occlusion, and possible temporomandibular joint dysfunction. These complaints are common early following surgery but frequently resolve. In fact, in some patients, changes in appearance may constitute an improvement. Because of expense and the perceived aggressive nature of the procedure, major facial-advancement surgery may be poorly accepted by patients with snoring or even mild-to-moderate sleep apnea. However, aside from tracheotomy, which bypasses UA obstruction, bimaxillary advancement surgery or aggressive mandibular advancement surgery demonstrate the highest surgical success rates.

Tongue Operations

Midline glossectomy and lingulaplasty are excisional procedures of the posterior tongue used for the treatment of tongue-base obstruction. They are speculated to enlarge the hypopharyngeal or retroglossal airspace. Midline glossectomy has demonstrated a 25% success rate in morbidly obese patients who had failed prior tracheotomy.[75,76] A more aggressive technique termed lingulaplasty demonstrated better results in a small series of 22 patients with a 67% success rate.[77] Most case series with these procedures are in obese patients with severe apnea, who have failed prior UPPP. Results of the studies show a dramatic reduction in respiratory disturbance. Success rates may be related to the aggressiveness of tongue resection. Similarly, complication rates are also directly related to the aggressiveness of the procedure.

New Surgical Approaches

Many new surgical procedures and technologies are being developed to treat OSAS. These include radiofrequency volumetric reduction of tissue, glossopexy, and electrical stimulation of the tongue.[78-80] These methodologies may offer conceptual improvements over existing techniques; however, no conclusions regarding their efficacy can be made at this time.

Tracheotomy

Tracheotomy bypasses the UA and avoids obstructions that occur during sleep. It reduces the morbidity and mortality associated with sleep apnea. The stoma can be plugged during the day to allow more normal function and appearance and opened only during sleep. Tracheostomy may be long term or temporary (ie, perioperative). The morbidity of tracheotomy precludes its wide spread use and includes risks of tracheal stenosis, infection, and altered appearance. For many patients, a tracheotomy is unacceptable. Because patients often have large necks, special tracheotomy techniques are required. These include several "skin flap" methods that allow improved and easier stoma care.

Perioperative Care

In the postoperative period, patients with OSAS have unchanged or worsened sleep apnea. Aggressive treatment to prevent airway obstruction is needed. This includes the use of perioperative nasal CPAP or tracheotomy and close observation.[81] In patients at risk for obesity hypoventilation, assisted

ventilation may be required. Close monitoring for airway obstruction, hypoventilation, and bleeding are required. Continuous pulse oximetry is frequently used but is an inadequate measure of ventilation or marker for intermittent apneas. This is especially true if a patient requires supplemental oxygen. Risks of respiratory problems increase with the combination of residual anesthesia effect, relocation from a stimulating environment (such as recovery room), sedation, dehydration with tenacious secretions, narcotic pain medication, and sleep onset. Close nursing observation is required and may warrant an intensive care setting.

Summary

OSAS is a disorder with an important structural etiology. Surgical procedures that effectively modify the upper airway may successfully reduce the respiratory and sleep disorders that occur with OSAS. No single procedure is applicable to all patients. For severe OSAS, surgery often requires applying multiple procedures directed at multiple airway segments. For less severe disease, a more limited, site-directed approach may be used.

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Discussion

Erman: It is important to emphasize that somnoplasty is FDA-approved at the moment for snoring, not for apnea, and there are no data that I'm aware of yet available to support its use in patients with apnea.

Woodson: Correct. There are no published data at this time. Somnoplasty is somewhat different from previous palate operations in that it does not acutely remove tissue. It may acutely make the airway smaller due to tissue edema. With LAUP (laser-assisted uvuloplasty) or other techniques that remove tissue, you may observe worse apnea. Airway area decreased about 8% in the one study using MRI.[1] With somnoplasty, however, you have a pharynx with redundant tissue, and a thermal injury, which can cause significant edema. In apnea patients (even those using nasal CPAP perioperatively), this is a potential problem. I think one has to be quite careful and think about exactly how one's going to apply somnoplasty to apnea. You do not want to apply it to everybody who walks in the door. I don't want to sound negative. Somnoplasty is promising, but we do have a lot to learn.

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Chediak: I had the opportunity to sit down informally with one of the investigators using this somnoplasty in February of last year (1996). He shared with me the preliminary results in patients with moderate degrees of sleep apnea. They do appear rather promising for sleep apnea. I think the unique feature of this 'toy,' as you correctly pointed out, is the ability to address various sites with one technology in a sequential manner. That, to me, is intuitively attractive as a means by which to structurally alter the upper airway.

Woodson: I think one of the things that appeals to me, and (as I teach my residents) for any kind of pharyngeal procedures, if you excise pharyngeal mucosa, you can't do anything but create a circumferential narrowing of the airway—the exception being when there's true redundancy. However, with a procedure like somnoplasty that preserves the mucosa, you may lessen that effect.

Erman: There is an allure to being able to pick the spot that you want to treat. The problem from a scientific perspective is, that despite numerous efforts by numerous people, trying various techniques and trying to predict at what site treatment should be given or what would be effective, few data support the hypothesis that there is any predictive value to assessments, measurements, or observations done in the waking state. Efforts to assess by looking at area of collapse, even in sleep, also have not been of great value in predicting what technique is going to be of benefit. Although it's alluring to say "I can treat at Site A or Site B," what we don't know is that this has good predictive value in terms of the outcome.

Woodson: Currently there are no methods to predict in a large number of patients how the airway's obstructing. We don't know if the airway behaves as a structure with different "zones" or as "one." The approach that I tend to take when I'm using multiple surgeries is that it's really one collapsible tube with areas of different compliance, under different degrees of neuromuscular control. If you make it large enough and stiff enough then you will prevent collapse.

Strollo: One of the things that the Stanford group is looking at for physical diagnosis is the arch of the hard palate and the intramaxillary and intramandibular distance which may have an impact on lateral pharyngeal wall and airway size. The problem in adults is being able to expand the maxilla appropriately and deal with that in a surgical fashion. It's a little easier to address in a pediatric population, but I think that it highlights that there are a number of different

defects in the upper airway that result in airway closure. Much of our surgery doesn't really do anything to address intramaxillary or intramandibular distance or the arch of the palate. As you know, Marfan's patients, who are thin, typically have that defect and have a high incidence of sleep apnea. What are your thoughts about the arch?

Woodson: A lot of people with apnea tend to have a maxillary retrusion. The face is long and narrow. The anatomic term is dolichofacial. With dolichofacial development the maxilla is often constricted.[1] This may also occur with a high arched palate. So, yes, I think the arch is an important marker. I'm not sure it has a direct effect on the airway, however.

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Strohl: You mentioned 3 techniques—the hypotonic technique, the multiple catheter technique, and then the P_{crit} or the determination of critical closing pressure—all of which could be performed at a sleep laboratory. While they may not have a lot of specificity for the area, they do give you a better handle than the apnea-hypopnea index as to how this unit is behaving. What role do they have, particularly with the resurgence in interest in surgical approaches? Should that be more routinely available?

Woodson: I think the most applicable technique is critical closing pressures. During a CPAP titration, one can determine the critical closing pressures. In normal individuals, the airway closes at negative levels of pressure, say, −5 cm H_2O pressure. People who are snorers are more around 0 cm H_2O pressure, and those people with the more severe apnea close in the range of +5, +10 cm H_2O. With a given patient with apnea and already at a +10, a minor intervention like a genioglossus advancement or palatal pharyngoplasty may not change the closing characteristics enough to provide an adequate clinical response. The same principle may be applied for a patient who is overweight. A 20-pound weight loss (which is a reasonable goal) is unlikely to change the apnea in a patient with a +10 cm H_2O closing pressure. On the other hand, if you did critical closing pressures and you get a level of +2 or +3 cm H_2O pressure, the patient might be a better candidate for limited surgery, palatopharyngoplasty, genioglossus advancement, or weight loss. A commercial triple-lumen balloon is being developed. The group ENT [ear, nose, and throat] in Antwerp has quite a bit of experience with this, and they believe that they are better able to predict surgical outcomes.

Atwood: Tucker, can you talk a little bit more about upper airway imaging—the different techniques. You showed a lot of cephalometrics. Cephalometrics are cheap. They're easy to get. What's your opinion about their role in planning surgery: Who should have them; should the pulmonologists be getting cephs ahead of time or are we wasting our patient's money? What about MRIs and other techniques?

Woodson: At this time, quite a few studies suggest that cephalometrics are not predictive.[1-5] They do discriminate among different groups, but as far as being able to predict outcome, they're unable to do so. Now we have a study[6,7] just recently published, that shows that part of the reason may be that there's a lot of 'noise' in the measures. There is so much variability in the population. You're not controlling for obesity, which has a big effect on how the airway collapses. Gender has a big effect. Age has a big effect on how the airway collapses. Then there are effects within the cephalometric x-rays themselves, of which probably the most important is skeletal facial type. In the study that we did, we looked at a group of Caucasian males, and we divided them by facial skeletal type. We actually found that there were a whole slew of measures that were predictive in identifying outcome, whereas, when we looked at the group as a whole, these measures didn't fall out. So, it may be important to actually stratify people by facial subtype, gender, age, and racial ethnicity to make cephalometric x-rays more useful. But at the current time I don't find them to be clinically useful.

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Atwood: A follow-up question. Do you get MRIs or is upper airway endoscopy better for you in planning surgery?

Woodson: For research I've gotten MRI scans. Right now I'm using endoscopy. It's interesting. In Vancouver, John Ryan is doing more objective endoscopy. He is putting a catheter down and then cor-

relating the size of the airway to the catheter. He feels he's getting fairly reproducible results. So it may be possible to do endoscopic evaluation in certain controlled circumstances and get reasonable numbers. We just did a study that looked at a variety of physical exam techniques and compared them. We found that posterior airway space on cephalometrics and endoscopic evaluation of the airway using my method correlated fairly well. So, maybe endoscopy is useful.

Strollo: As a sleep doc and pulmonologist who does a lot of cephalometrics and is involved with a surgical program, I think one of the benefits of cephalometrics is for planning skeletal surgery and choosing patients, whether they're going to go to Phase 1- or Phase 2-type approaches. If your approach has been that everyone gets a Phase-1 approach, then you can question your decision making, but it is important for the surgeon to know what the alignment of the teeth is and how those structures are going to fit together. What we do at Pittsburgh is share our cephalometrics when we do them. If you're going to treat somebody medically, there's probably no role for cephalometrics in terms of prediction. One of the things that I think has clouded the literature on uvulopalatopharyngoplasty is that we don't routinely separate out tonsillectomy from palatal procedures, and the value added of a palatal procedure on top of a tonsillectomy is frequently hard to tease out. Some work done by our group[1] showed that when the tonsils are not present, which may impact on lateral walls, your chance of success is about 7% as opposed to the typical 50%. What are your thoughts on that, Tucker?

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Woodson: The Pittsburgh group has published a couple of papers saying that if you take out the tonsils people do better, but it's just a small series right now. It may be true, but there's also a small series saying that it's not true. I think in that group of people who have RDIs > 50-60, the presence or absence of tonsils may not be very predictive. However, the group of people who have RDIs of 20-40 or 20-50, tonsils may be more predictive. This may be why the literature is different. That's my gestalt—that there are no data.

Sleep-Disordered Breathing in Children

Lee J Brooks MD

Introduction
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Summary

Introduction

Children with obstructive sleep apnea (OSA) share certain clinical features with adults, but important differences are seen in the clinical presentation, diagnosis, and treatment that suggest that, although the pathophysiology is similar, it is probably not the same.

Among children, 12% snore "on most nights," and significant sleep and breathing disorders occur in almost 1% of 4-5 year olds.[1] OSA can have important effects on daytime cognitive function[2] and behavior[3] and on cardiopulmonary function, so prompt recognition and treatment is imperative.

Although the clinical appearance of children with OSA is often different from adults, in both groups the problem is usually recognized by the female caregiver (wife or mother), and there is often some denial and reluctance on the part of the patient to seek medical care.

Predisposing Conditions

A significant fraction of children with OSA have some obvious underlying defect. Almost one third of Brouillette's patients had obvious facial deformities, such as those present in Pierre-Robin syndrome and Crouzon disease, likely to reduce pharyngeal size.[4] Of course, pharyngeal size cannot be the sole factor in the development of OSA; if it were, both children and women, with their smaller pharynxes,[5] would have a higher incidence of OSA than men do. In Guilleminault's description of 50 children with OSA, 16 (32%) had facial dysmorphia, but an additional 6 (12%) had underlying neuromuscular disorders, probably causing OSA through pharyngeal hypotonia.[6] Children with Down syndrome[7] or Prader-Willi syndrome may be at particular risk for OSA because the problems caused by an anatomically small pharynx can be compounded by decreased neuromuscular activation.

As in adults, obesity may predispose the child to OSA.[8,9] In contrast to adults, however, children with OSA may fail to thrive. Of the children in Brouillette's series,[4] 27% were failing to thrive, probably because of increased work of breathing during sleep.[10] Such children usually display catch-up growth after effective treatment for their OSA.

Clinical Presentation

Although the apnea occurs during sleep, its effects can manifest during wakefulness as well. Nocturnal symptoms include snoring, which is present in virtually 100% of children and adults with OSA. Often, respiratory pauses have been witnessed by a parent. In children, it is important to differentiate central apneas (without respiratory effort) from obstructive events. Some central pauses can be normal in young children, but apneas that produce bradycardia or oxyhemoglobin desaturation may imply a disorder of respiratory control. Children with OSA may be described as struggling to breathe or sucking in the chest. Other nocturnal symptoms

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include disrupted sleep with or without nightmares and nocturnal enuresis inappropriate for age.

Daytime symptoms are usually those suggesting sleep deprivation. Nocturnal respiratory events often result in frequent arousals, but these arousals may not be apparent without electroencephalography (EEG). Nevertheless, these arousals serve to disrupt the patient's sleep architecture resulting in a lack of refreshing sleep and symptoms of excessive sleepiness during the day. Adults may describe forgetfulness or falling asleep at work or while driving.[11] Children may simply manifest irritability, hyperactivity, aggressive behavior, or discipline problems. Learning problems have been known to be a manifestation of OSA for over 100 years, since Dr Hill noted "the stupid looking lazy child who frequently suffers from headache at school, breathes through his mouth instead of his nose, snores and is restless at night ... is well worthy of the solicitory attention of the school medical officer."[12] Early morning headaches may be a result of CO_2 retention. Long-term sequelae include polycythemia to increase oxygen-carrying capacity in the face of hypoxemia and systemic hypertension. Right ventricular hypertrophy or cor pulmonale are reported in 27-56% of patients,[4,6] probably a result of hypoxemia and/or marked fluctuations in intrathoracic pressure during obstructive events.

Laboratory Evaluation

Although history and physical examination are important in suggesting OSA, the diagnosis must be confirmed by overnight polysomnography (PSG).[13] Fewer than half of the patients who are referred to a sleep disorders center with symptoms suggesting OSA actually have the disease, despite snoring, reported apneas, daytime somnolence, and similar complaints.[14] In this study, the diagnostic accuracy of the subjective impression formed by a specialist who examined patients referred because of suspected OSA was only 63%. A retrospective study of children revealed that only 37% of children referred because of suggestive symptoms actually have OSA.[15] Prospective studies suggest that only about half of children presenting with a clinical history suggesting OSA, including some combination of snoring, respiratory pauses, and daytime sleepiness, actually have the syndrome.[16,17] Brouillette et al[18] devised a clinical score that was both sensitive and selective in identifying children with OSA, but this has not proven effective when tested prospectively in other populations.[16] Augmenting the clinical history with videotaping (ie, a 'sleep tape') resulted in a positive predictive value of only 0.5.[19] A limited home polysomnogram combined with videotapes was successful in identifying selected children with OSA,[20] but these results have not been confirmed in a larger population outside the research setting.

The best test to confirm the diagnosis of OSA remains the polysomnogram. This all-night study usually includes EEG, electro-oculography, and chin electromyography (EMG) to monitor the stages of sleep; determination of airflow at both the nose and mouth, usually by thermistors or capnography; assessment of chest-wall and abdominal motion by inductance plethysmography or strain gauges to assess "paradoxical" breathing and differentiate obstructive from nonobstructive events; electrocardiogram; oximetry; and pretibial EMG leads to detect motion.[21] More limited studies may be adequate if central apnea or gastroesophageal reflux are the sole concern but such studies are inadequate to diagnose OSA with confidence. In addition, many nonpulmonary disorders can produce excessive sleepiness, including nocturnal myoclonus and narcolepsy, which may exist independently or in conjunction with OSA.[22] PSG is the best laboratory tool to distinguish the many causes of excessive sleepiness. Expertise with children is particularly helpful because patience and a pleasant environment are essential to obtaining quality data.

Normal PSG values in children are not well defined. Normal young adults have fewer than 5 respiratory events per hour of sleep (respiratory disturbance index, RDI),[22,23] and this threshold has been applied by several investigators. Men with an RDI > 20 may have greater mortality than patients with an RDI < 20.[24] The elderly tend to have more frequent respiratory events, and infants and children may have fewer.[25] One study[26] suggests that > 1 obstructive apnea per hour of sleep is abnormal in children. This study did not consider hypopneas or central apneas following movement nor was there EEG confirmation of sleep. No data define any clinical consequences of so few respiratory events. Because young patients may not exhibit the classical obstructive pattern seen in adults,[27] the laboratory must be experienced with children in order to detect these often subtle events. The number of arousals and extent of oxyhemoglobin saturation must also be considered in determining the severity of disease. Non-sinus cardiac dysrhythmias are uncommon in children, but when present indicate the need for prompt intervention.

Although adenotonsillar hypertrophy is commonly associated with OSA, the size of the tonsils and adenoids do not predict the number of nocturnal respiratory events.[9] The PSG is also helpful in predicting response to treatment and the need for follow-up,[17] as well as predicting postoperative risk and the extent of monitoring that is required after treatment.[28,29]

Seasonal variations in symptoms suggest the possibility of an allergic component, which can be defined by intradermal testing. A clinical trial of nasal decongestants (Afrin™, neosynephrine) and/or anti-inflammatory medications (nasal steroids or cromolyn) may be warranted. Other studies that may be useful in evaluating selected patients with OSA include hematocrit to detect polycythemia; arterial blood gas analysis to assess hypoxemia and hypercarbia; electrocardiogram and/or echocardiogram to evaluate the possibility of right heart strain; and chest roentgenogram. Thyroid function testing is necessary in individuals whose clinical presentation suggests hypothyroidism. Pulmonary function testing may define any underlying lung disease that may predispose to hypoxemia or demonstrate the decreased inspiratory flows and "saw-toothed" flow-volume loop that may be seen with upper airway obstruc-

tion.[30] Cephalometric radiography may help distinguish subtle bony abnormalities that may predispose to OSA.[31] A simple lateral neck film can be useful for assessing the impact of adenoidal hypertrophy on the pharyngeal air column. We often use the acoustic pulse technique to assess pharyngeal size and compliance,[5,32] but this may not be available in all centers. Computed tomography,[33] magnetic resonance imaging,[3] and direct visualization by laryngoscopy have also been suggested to image the pharynx and may be useful in determining the best treatment option for an individual. The choice of these ancillary studies is dictated by the patient's clinical presentation.

Sequelae

Left untreated, obstructive sleep apnea can have serious sequelae in children. As mentioned earlier, disruption of nocturnal sleep can cause irritability, hyperactivity, aggressive behavior, or discipline problems. Learning problems have been known to be a manifestation of OSA for more than 100 years.[12] Hypersomnolence may place teenage drivers at increased risk for auto accidents.[11] Early morning headaches may result from CO_2 retention. Long-term sequelae include polycythemia to increase oxygen carrying capacity in the face of hypoxemia and systemic hypertension. Cor pulmonale may result from hypoxemia and/or marked fluctuations in intrathoracic pressure during obstructive events[35] and has been reported in about one fourth of children with OSA.[4] In some series, over half of these children fail to thrive.[6] Untreated men with severe OSA and a respiratory disturbance index > 20 have a significantly greater risk of mortality than do patients with an RDI < 20.[24]

Treatment

Treatment of children with facial dysmorphia or underlying neurologic disorders is best accomplished by an interdisciplinary approach aimed primarily at the underlying illness.[36]

Factors to be considered in deciding on the vigor of treatment include polysomnographic measures, such as the RDI and extent of oxyhemoglobin desaturation, and clinical factors, such as daytime sleepiness, school performance, and behavioral difficulties. Intervention may be suggested in borderline cases in which the severity of the patient's snoring is interfering with normal family dynamics.

Treatment strategies can be divided into behavioral, medical, surgical, and nasal continuous positive airway pressure (CPAP). Many children simply require behavioral modification, including weight loss and avoidance of alcohol and sedatives (commonly found in over-the-counter cold preparations) at bedtime. Respiratory stimulants such as progesterone[37,38] or nicotine[39] have had limited success in adults and are rarely indicated in children because of potential side effects. Protriptyline may be helpful in selected cases by decreasing rapid eye movement (REM) sleep[40] or by selectively stimulating pharyngeal muscles.[41] Although oxygen can reduce hypoxemia it does not reduce apneas or improve daytime hypersomnolence[42] and should rarely be the sole treatment. Dental appliances have been devised to reposition the mandible, shift the tongue forward, or otherwise enlarge the pharyngeal air space. However, adequate data are not yet available to prove their continued long-term efficacy.[43]

Surgery remains the most common treatment for OSA in children. Selection of the procedure must take into account the child's potential for growth. Even though the craniofacial skeleton has attained 60% of its adult size by age 4 years, facial growth continues until about age 16 in girls and age 19 in boys.[25] The risk of altering facial growth by surgery must be considered, especially because changes in facial structure with growth may lessen the severity of the syndrome.

Tracheotomy can cure virtually all cases of OSA by completely bypassing the upper airway, resulting in a marked reduction in mortality.[24] However the morbidity of tracheostomy is not insignificant,[44] especially in young children, in whom it can adversely impact psychosocial and language development.[45] Procedures such as mandibular advancement and hyoid resuspension have been successful in selected adults with OSA,[45] but many surgeons are reluctant to perform these procedures on children whose facial structures are still growing and changing.

Uvulopalatopharyngoplasty (UPPP) is an excellent surgical treatment for snoring; however, only about half of patients have a significant improvement in apnea.[47] Although patients with a good response to UPPP tend to have a smaller pharynx than those with a poor response,[33,48] considerable overlap exists between the groups, and it remains difficult to determine a priori whether an individual will respond.

Adenotonsillectomy remains the oldest and most common means of treating OSA in children and is effective in as many as 90% of the patients without craniofacial or neurological abnormalities.[6] It can result in prompt remission of cor pulmonale[11,49,50] and improvement in school performance.[12] However, it does not affect central apneas or hypopneas,[5] and it appears that patients with the most severe disease do not respond as well as patients with a more mild condition.[17] A baseline PSG is essential to determine severity in the individual patient and the likelihood of a good response to surgery. Lymphoid tissue changes with age, and mild OSA may resolve spontaneously with normal regression of the tonsils and adenoids. Conversely, OSA may reappear several years following apparently successful adenotonsillectomy.[25] Children with severe OSA at baseline and those with persistent symptoms should have overnight PSG about 1 month after adenotonsillectomy to ensure resolution of their apneas.[17] It is also wise to observe the patient closely in the immediate postoperative period because the combination of sedation and surgical edema may exacerbate obstructive events. Patients who are young (< 3 years of age), who have craniofacial abnormalities or other medical problems—such as cor pulmonale or morbid obesity—or who have severe OSA confirmed by PSG require close observation as inpatients following adenotonsillectomy.[28,29]

In patients without adenotonsillar hypertrophy, patients with an inadequate response to adenotonsillectomy, or patients who would prefer to avoid surgery, nasal CPAP is a useful option.[51] This so-called pneumatic splint results in prompt improvement in cardiac function,[52] and in adults results in cure rates superior to surgery.[53] It has been employed successfully in children with craniofacial abnormalities[54] and in otherwise normal children as young as 15 months.[55] With proper education of patients and their families, excellent long-term compliance can be achieved.[56] CPAP may be particularly useful while other long-term treatments such as weight loss are being implemented.

Determining the level of CPAP required for the individual and the type of mask that provides the most comfortable delivery of pressure is best done in a sleep laboratory experienced in CPAP titration in children. We do not recommend attempting to titrate CPAP in the home or in the latter half of a diagnostic PSG because of the education and manipulations required. Children may require changes in mask size or pressure as they grow, so regular follow-up with an individual experienced with OSA in children is essential. Unlike adults, children may be able to discontinue CPAP with growth of the facial structures and/or maturation of neuromuscular function of the pharynx.

Summary

Almost 10% of children who snore have significant sleep and breathing disorders. Obstructive sleep apnea results in nocturnal hypoxemia and sleep fragmentation that can lead to poor school performance, behavior problems, hypertension, cor pulmonale, and failure to thrive.

Treatment should be considered only when the severity of the syndrome has been established by objective testing. Overnight polysomnography in a laboratory experienced in the evaluation of children remains the gold standard for differentiating primary snoring from OSA. PSG can also guide the choice of treatment, intensity of monitoring, and need for follow-up. In otherwise normal children with OSA and adenotonsillar hypertrophy, adenotonsillectomy is usually curative of mild-to-moderate OSA. In children with more severe OSA, nasal CPAP may be required. CPAP is also helpful in children with neurologic problems or craniofacial problems in whom surgery is not yet appropriate. CPAP is best initiated by an individual with skill and experience with CPAP in children.

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Discussion

Woodson: Pediatric sleep studies confuse me. An RDI [respiratory disturbance index] of 1 or 5 defines the level of pathology. I think that most people in the room would say that arousing once or even 5 times a night is not pathologic. I would assume that the RDI is, actually, a marker of underlying hypopnea and upper airway resistance. I think the pediatric population is the ideal population for the upper airway resistance syndrome. Does using traditional, even modified, adult criteria applied to children miss the disease? My concern with polysomnography in kids is that it's not directly measuring their disease. Do other methods

need to be used, such as objective flow sensors or some measure of esophageal pressures? Now, obviously, you're not going to put a balloon or a catheter down a kid, but things like pulse, transit time, or other measures actually could pick up the disease in kids. Calling an RDI of 1 pathologic just, to me, doesn't make sense. Could you comment on that?

Brooks: Sure, we actually have 2 points there. One is What is the meaning of an RDI of 1? I tried to make that point. That was the statistical cutoff in one single study that looked only at apneas.[1] We talked about some of the problems with that study. I personally don't think that an RDI of 1 or 2 puts someone at a lot of risk for serious complications. I actually have some data that show that because we have followed up the kids in our study (in which we considered an RDI of 5 a cutoff). Because we decided not to treat that half of kids with an RDI < 5, we were concerned "Gee, are we missing something? Are these kids going to come back in a couple of years in cor pulmonale?" We brought those kids back, and they were no more severe than they had been earlier, and they did not have signs of any other sequelae. So, I agree with you that 1 is a statistical cutoff based on a single study, but I personally have a higher threshold for treating than that. Regarding the point about missing things, I don't think we are. We have a lot of measures of air flow. Two of them are direct, the thermistor and the capnograph; and one of them, the inductive plethysmograph, is indirect. I really think that not many kids who go through my lab and come out as normal really have the upper airway resistance syndrome. I think that you tend to see that more, if you'll forgive me, in an adult-oriented lab where, when your average patient may have 50-60 clear obstructive apneas, you may not be looking as hard for the more subtle events. But I think if you look for them, they're there.

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Woodson: I think that most people want objective testing, but that there's a huge amount of clinical experience out there that shouldn't be ignored. A lot of these kids who have "negative sleep studies" clinically improve after tonsillectomy and adenoidectomy. We see growth spurts; we see behavior changes. Many pediatric otolaryngologists don't rely heavily on polysomnography. It would be nice to explain this dichotomy. I think it would improve patient care all around.

Brooks: I don't think it really is a dichotomy. First of all, we can all get ingrained into treatments without testing whether they help. For decades we were treating kids with cystic fibrosis with mist tents because we thought they helped; and, eventually, somebody did the study and showed that they don't mean a 'hoot in hell,' so we don't use them anymore. I hope that nobody bases their decision for treatment solely on a laboratory test, no matter how well performed. Certainly, the more severe the clinical presentation, the more likely we are to recommend treatment for a given level of severity in the lab. If a patient has an RDI of 4 and the parents say that he snores but he's active during the day, he's doing well in school, he's not a behavior problem, [and] he doesn't seem sleepy, I probably wouldn't treat that kid. But the same patient who has an RDI of 4 who is falling asleep in school and has some problems, then I would treat him. I think that for adults as well as kids, you look at the patient as well as the laboratory. The two together help you make your decision on whether to treat and what type of treatment.

Chediak: I wonder if perhaps the lower numbers of respiratory abnormalities in children as the statistical cutoff point you showed us is related to the resting respiratory rate. For example, adults, when breathing normally and not through a mouthpiece with the nose occluded, will have an average respiratory rate of about 16 breaths/min while awake. So that if you have a 10-second apnea then what you're saying is that the average respiratory rate would be 6 breaths/min, if 1 breath is separated from the next by 10 seconds or more. Now, children have much higher respiratory rates. Shouldn't they have a lower cutoff point for duration of apnea?

Brooks: Great question and there are people who do apply a different criterion, which would be that 2 missed breaths implies an apnea. Well, if you have a baby who's breathing at 60, 2 missed breaths is just a couple of seconds, and I don't think that has any clinical significance. There isn't an answer here. Like I said the other day, 10 seconds is because we have 10 fingers. I personally find it very difficult to constantly be measuring the respiratory rate and figuring out how long an event should be, without any real evidence that it matters. So, right now, I'm opting for the simple approach of picking an arbitrary number of 10, which you seldom see, even in infants. You seldom see a major desaturation or a major bradycardia with a 10-second pause. So, I think that from a clinical point of view we're safe with 10. That also gives us the advantage of being better able to compare number of events because we're not at a moving target, either within an individual or between individuals. But your point is well taken, and there are people who disagree with my approach.

Minkley: I wonder if the challenge put out by Dr Woodson might be directed to registered polysomnographic technologists, where specialists like you trained in pediatrics as well as pulmonary and sleep medicine and surgeons like Dr Woodson trained in both aspects do not exist. These broadly-trained people aren't available to us routinely. The sleep center may be the clearing house for these patients—may be the place where you have a technically oriented staff who also have clinical knowledge

and can at least direct patients in some way based on multidisciplinary decision trees. A patient assessment might help decide the correct level of testing for a better workup, for better treatment. But if it's as we've seen here—if treatments and assessment involve multidisciplinary areas and there's no coordination, I wonder what might be happening to people, particularly kids.

Brooks: I don't think kids are really different in this respect. I think that a multidisciplinary approach is helpful for any age. We've heard about treatment options ranging from CPAP to medications to surgery. Tucker (Woodson) just gave us a dozen different surgical procedures applicable to selected adults. So, there's always a place for a good multidisciplinary clinic where you have the sleep expert (who may or may not be a pulmonologist) and a surgeon. You may want an ENT [ear, nose, and throat] surgeon and an oral surgeon, for example. I think that's a good idea if it can be arranged logistically.

Strollo: There have been a couple of notions in the adult literature that adult sleep apnea is really a consequence of pediatric problems, such as breathing against an increased resistance for a period of time, not doing a tonsillectomy or doing a late tonsillectomy, influencing craniofacial growth, allergies in childhood—which may alter upper airway anatomy—and also in kids who have craniofacial problems or have a high arch. At least the Stanford group seems to be in favor of using functional orthodontic appliances to help redistribute mandibular growth. How do you see that whole issue shaping up in the pediatric world?

Brooks: Those are really hard things to study retrospectively. They're even harder to study prospectively. Certainly what we do in childhood affects what happens in adulthood, and that's why most of us are very hesitant to use things like oral devices and some of the jaw surgery that we heard about earlier. When I'm paying thousands of dollars to an orthodontist for my kids, I don't know what 'monkeying' around with their pharynx is going to do. Are we going to have to do that [orthodontia] again? What's going to happen here? I guess right now I'm watching that stuff with interest, but I'm not ready to apply those types of things. There is also a tendency to pick out small groups of patients and then make great sweeping statements about the results. Stanford has a lot of experience going back for more years than many of us, and they've come up with some good ideas, but, in my mind, they're not all correct.

Strohl: To what degree are our current treatment strategies for children based on clinic-referred patients? That is, is there a bias in terms of either our approach or our strategies because current knowledge is based on what people refer to us? Where do you see us going once we get more information about what's out there in the community? The flip side is that many of the patients have either craniofacial abnormalities or tonsillar hypertrophy that have been reported in clinical surveys of OSA and the family. That's not the major cause or major association in the community-based surveys to date.

Brooks: I think that with any type of subspecialty clinic you're generally seeing the tip of the iceberg. Many kids who snore are referred directly to a surgeon who takes out their tonsils and adenoids—and that's it. So, we probably do get the more severe and maybe even the more educated, but I think that that's true about any of us, as subspecialists. What was the rest of your question?

Strohl: I think the community-based studies will get us to think about prevention and early detection—the finding that there are children as well as adults out there who don't meet the cartoons that we've developed from our clinic-based practice, that is, they're not obese, they don't have big tonsils, they don't have craniofacial abnormality, they aren't the caricatures that we think of. Then there are those who have many apneas and are asymptomatic altogether. I think that gets us to think about prevention and prevalence and how disease and illness are expressed. That's where I think the adult literature is going, and I think you're at the same point that perhaps we were 6-8 years ago, before we had epidemiologic studies that made us rethink what we're doing in the specialty clinic.

Brooks: One key difference between kids and adults, though, is that kids are growing and changing. Their faces change up until the teenage years and their lymphoid tissue is going to get bigger and then regress some. Unless you're thinking of preventing disease in later life, I don't know how we would study prevention except with longitudinal studies, which are hard to do.

Strohl: The advantage of kids over adults is that a 5-year prospective study of a group of kids gives a lot more insight into the changes in physiology that might happen with changes in airway size and airway compliance and anatomy than would a 5-year study in a 50-year-old adult population. So, I think even though it's more complicated, the opportunity exists to really get some insight into this. The other is to what extent did the sleep habits of the family, sleep habits of a community impact upon the behavioral sequelae? The evidence is that for familial patterns of sleep, sleep length, that a lot of that is under very tight parental control in those age groups. Concordance of sleep patterns between twins who are reared apart is much better as they get out of the home than when they are within the home—again, twins reared apart. It's the attitudes of their parents, and therefore, their sleep length, other things that impact upon the behavioral things that we talk about often in kids rather than the cardiovascular problems.

The Impact of Managed Care on the Practice of Sleep Disorders Medicine

Milton K Erman MD

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Introduction

Tremendous changes have occurred in recent years in the health care and medical service industries of the United States. Foremost among these have been the increasingly important roles that managed care delivery systems and entities have come to play in the health care system in the United States. The impact that these entities have had on the practice of medicine and the organization of health care delivery models has been enormous, influencing reimbursement rates for various services and procedures and at times redefining concepts of what is appropriate care for various disorders. In many parts of the country, dramatic reductions in rates of hospitalization and in lengths of stay once a patient is admitted are testimony to the power that is wielded by these payers.

The degree to which managed care influences or 'controls' the medical marketplace varies from region to region across the United States. However, it is clearly the case that in all parts of our country these entities play an important role—a role likely to be increased in the future—in defining specific delivery care models for patients with a broad range of conditions and disorders. These models have already influenced diagnostic and treatment patterns for patients with disturbances of sleep and breathing, and there is no evidence as yet of a reversal of the trends of growth seen in managed care, almost all of which are at the expense of the traditional fee-for-service system. I believe that to deny this reality or to believe that 'problems' with managed care companies can be avoided by not dealing with them is foolish and misguided.

To understand the impact that managed care has had on sleep disorders and sleep-related services and, possibly, to have an impact on decisions made by providers about care for patients with sleep disorders, we must understand the forces that have stimulated the development of managed care plans and the factors that continue to promote growth in these plans. We may also benefit from understanding concerns that have been expressed regarding managed care systems and the quality of care they provide, potential conflicts of interest they may have with regard to care they provide, and barriers that may interfere with the receipt of care by their patients.

Origins of the Health Care Funding Crisis

The forces that have nurtured the development of managed care in the United States are clear and obvious. The

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cost of delivery of health care has increased dramatically over the last 4 decades, by any measure utilized. Health care expenditures had risen to almost a trillion dollars ($949.4 billion) by 1994. Stated as a percentage of gross domestic product (GDP), this reflected an increase from 5.1% of GDP in 1960 to 13.7% in 1994. This figure of close to 14% is roughly twice the proportion of GDP spent on health care in other developed countries, without measurable differences in national health indices such as longevity and infant mortality.[1]

How and why did such a system, which generated grossly disproportionate expenditures of resources on health care, become established in the United States? Although many factors may have contributed to the growth of the system, a major element in the development and maintenance of our bloated health care establishment has been the fee-for-service reimbursement structure that had always been the payment mechanism in the United States. Reimbursement in fee-for-service systems is procedure- and encounter-driven. The practitioner is rewarded for providing care; the patient is gratified to be receiving "the best care possible," to know that care will include "all that can be done" without concern for the cost of any single procedure or the total share of national resources that health care is consuming. Other factors stimulating health care expenditures have included an overvaluation of the role that new and expensive technologies could play in virtually all practice areas, profits as an incentive for hospitalization and provision of services, and reimbursement systems for medications and procedures that tried to ensure that treatment choices were not inappropriate, rather than the ones defined as most appropriate and cost-effective.

Prior to the establishment of essentially universal insurance coverage for full-time workers and their families during and after World War II, health care expenditures were constrained by the financial limitations of the consumer. To obtain care, services needed to be paid for by the patient or family. Health insurance coverage, which usually paid all or virtually all the costs for health care defined as "reasonable," removed financial constraints as an element that had limited utilization of costly medical resources. The growth of the Medicare program, established in 1965 on a fee-for-service basis, contributed to this trend as well, with essentially absent oversight or utilization review of costly procedures for the elderly, such as cataract surgeries, cardiac angiography and bypass procedures, and home health services. State-funded programs for the medically indigent have lead to similar increases in utilization and in costs associated with treatment.

Mushrooming health care costs spurred the development of new service and technical industries to provide more aggressive and more comprehensive care across all strata of society. Hospital staffs and hospital bed counts grew; medical school class sizes were increased, and new medical schools were opened to provide adequate numbers of physicians to cure society's ills. False economic 'productivity' was generated by such activities, without clear evidence that the revenues expended benefited society or reflected a rational use of resources. The availability of these services without constraints and at little or no out-of-pocket cost to the patient generated a false sense of security for the consumer, who enjoyed access to increasingly sophisticated and costly services but did not feel the need to claim ownership responsibility for the costs associated with provision of these services.

Who paid the burgeoning health care costs associated with this-fee for-service, technology-drive system? The lion's share came from employers, including local and state governments and from the federal government in its role as employer. The impact of this growth curve on all employers and payers was clear: massively increased expenditures to provide health care for employees and retirees, without obvious or tangible benefits to the employer or to society as a whole. Employers were thus receptive to suggestions that alternative models for provision of medical care be considered.

Alternative systems that provide solutions to some of these problems of health care delivery have existed for some time. One novel approach was the development of prepaid health care plans, such as Kaiser Permanente Medical Programs and the Group Health Cooperative of Puget Sound on the West Coast, and the Health Improvement Group in New York. These programs, which were founded in the 1930s and 1940s, provide comprehensive health care for their members in an environment free of fee-for-service pressures. They emphasize preventive care and health screening, and often utilize health care extenders, such as physicians assistants and nurse practitioners, to perform triage functions and routine health maintenance activities provided by physicians in traditional delivery system models. The economic pressures that have become so evident over the past 25 years have caused these programs to flourish.

In 1973, the federal government of the United States began providing support for the development of health maintenance organizations, or HMOs. This reflected a conscious effort by Washington to try to control burgeoning health care costs and to effect change in systems for health care delivery. Employers recognized an opportunity to reduce costs and improve profits, and responded by providing incentives to employees to select HMO or preferred provider organization (PPO) plans. In some areas (such as California) where major growth of HMOs occurred, some employers even eliminated traditional (but costly) indemnity insurance as a health care coverage option that employees could select. To what extent have HMOs changed the traditional relationships and alliances that had existed in American medicine? HMO enrollment grew from 29 million in 1987 to more than 45 million in 1993; PPO enrollment grew from 12 million in 1987 to more than 77 million in 1993.[2] As a consequence of these forces, two thirds of Americans working for medium- and large-sized companies are now enrolled in managed care plans.[3]

The impact of changes in insurance coverage on retirees has been even more dramatic than that on the working population. In 1988, 60% of firms with 10 or more employees offered health care benefits to retirees age 55-64. The proportion declined to 46% in 1993, and to 40% in 1996. If that trend continues, it is projected that only about 1 in 8 workers age 40-45 today will be offered any health care coverage by their employers if they choose to retire before age 65. This has created a new health care crisis for early retirees, who must face a gap in their insurance coverage as they await their 65th birthday and initial eligibility for Medicare coverage. It has also made health care coverage other than Medicare for retirees over age 65 an increasingly rare commodity.

In an editorial in a recent issue of the *New England Journal of Medicine*,[4] Jerome Kassirer, commenting on an article by Eli Ginzberg, a prominent health care economist, noted that the origins of today's health care financing crisis were recognized decades ago by Ginzberg. In 1959, Ginzberg suggested in testimony before Congress that plans to enlarge the physician work force would create a surplus of physicians without improving the health of the public. He later predicted the growth of increased spending without commensurate improvements in quality of care, accurately predicted the growth curve for managed care, and noted that it was possible to control costs *or* to improve quality but not to do both at once.

In his article,[3] Ginzberg notes the substantial growth in size and influence that the HMOs have attained in recent years, but he also expresses his opinion that HMOs will not be able to continue to grow at a rate comparable to that of recent years. He points out that reductions have occurred in corporate costs for health care benefits and in the growth rate of national health expenditures but notes that HMOs may face new, perhaps insurmountable difficulties in further reducing the cost of care delivered or in providing care at all, to some currently underserved populations, especially those patients now on Medicaid.

Efforts have been made in a number of states and localities to regulate HMOs and to restrict the degree to which certain cost-saving measures may be implemented. For example, regulatory restrictions have been put in place preventing the "premature" discharge from hospital after delivery of healthy mothers and their newborns, even if no valid medical reason for continued hospitalization exists. The gap between expectations and results that patients have begun to experience in HMO settings may also lead to reductions in HMO enrollments in the future. Patients often express resentment at restrictions in selection of primary care physicians and limited access to specialists.The need to obtain authorization from their health plan or a referral from a primary care physician (who is encouraged to restrict 'costly' referrals) to see a specialist may influence patients to consider care alternatives that are more costly but which allow greater freedom. Employers in competitive marketplaces may also find that offering more comprehensive health care options may help them to more readily hire and retain qualified employees.

Capitation: Through the Looking Glass

Capitation provides one financial and organizational model for delivery of health services. In a capitated model, a fixed amount of money is available to a system, institution, or medical group to provide all necessary care for a specified period of time for a specific patient population under contract.[5] Some money is retained to pay for costly hospitalizations, which must be kept as brief as possible to limit expenditures for inpatient care. Some money is needed to administer the plan, to pay for prescriptions and other medications, and to pay for contracted services that may not be available within the plan or at a site convenient for patients (ie, physical therapy, emergency care, and some subspecialty services and procedures). What is left can then be divided among those who will actually provide care for the group, using some allocation model for service delivery that is likely to be arbitrary and unfair to some specialty areas.

Most physicians and other skilled health care providers have spent their entire professional lives functioning in fee-for-service environments. As a result, the implications and imperatives associated with service provision in managed care models, especially those which are capitated, may often be difficult to grasp. Relative to the classic fee-for-service model, in managed care settings the world is turned upside down. Revenues are no longer tied to or generated by procedures and patient visits; rather, they are assumed to be a resource available in a scheduled and predictable manner for the provision of all health care needs of the members of the plan. Billings, procedure volumes, and apparent profits are of less concern when a medical service or program is perceived as a cost center, rather than as a profit center.

In this setting, the financial and operational goals for a medical service program or department may include reductions in total costs associated with the provision of all health care services. The service that is provided is a drain on the system; the problem that is not addressed generates cost savings and improves the 'bottom line.' To those who have spent their careers providing medical care in fee-for-service settings, the adjustments in expectations and behaviors required to function in managed care settings may evoke feelings reminiscent of those of Alice in the Lewis Carroll classic, *Alice in Wonderland.*

Revenue does play a role in managed care settings, but in a fashion that is also altered by its trip "through the looking glass." In a capitated model, resources are usually not allocated to providers based on the amount of care that is provided. Instead, revenues are distributed, at least in part, based on models that attempt to define the volume of care that should be provided. Although efforts to make use of scientific methodology can be made, of necessity arbitrary elements will be

incorporated in such models, influencing how revenues are actually allocated. Using revenue allocation models and actual figures for the costs of services provided, balance sheets are created to determine whether the revenues credited to a division as a result of the allocation scheme will pay for the services provided. The revenues involved are fixed, having been determined by the allocation model and the number of patients being cared for. If a revenue shortfall is perceived to be present, cost savings must be accomplished or alternative revenue sources found.

One way to attain cost savings is by making reductions in the overall cost of services provided. Such savings may be effected in many ways. In the specific case of sleep disorders medicine, savings occur when there are reductions in the volume and/or unit costs of sleep studies performed. This may be accomplished through contracting with an outside provider for services at a reduced rate and through utilization review, which is intended to, and which does, delay provision of or reduce the volume of services provided. Costs may also be reduced by reductions in total staff numbers, or by the replacement of experienced, more 'expensive' employees with cheaper 'new hires.' Research and training are inherently expensive and 'inefficient' activities (ie, activities that do not serve the institutional purpose of providing quality care to a defined patient population at the lowest possible cost); even if secure sources of funding are available for these activities, they may be too out of step with the rest of the institution to allow their continuation. Finally, if the perception is that costs exceed allocated revenues, the sleep disorders center may be closed or 'out-sourced,' with the administrator (rather than the Queen of Hearts) crying out, "Off with their heads!!" The stresses associated with provision of care in such environments was addressed in a recent article in the *Journal of the American Medical Association,* titled, appropriately enough, "Capitation or Decapitation: Keeping Your Head in Changing Times."[6]

Econometrics & Sleep Medicine

Sleep Medicine in the Managed Care Environment

Economic models can be developed that incorporate the expected costs of providing care to patients with any type of medical problem and the projected savings generated by such activities. An econometric model of this type has been generated by Stephen Johnson, a practicing neurologist and managed care administrator, who also has formal business training, including a master's degree in business administration. Johnson, stimulated by his experience as a fellow in sleep disorders medicine at Scripps Clinic, developed an econometric model, dealing with issues specific to sleep disorders medicine.[7] Although the model was not based on the actual size of the Scripps population, it attempted to define the true costs of provision of service and the projected cost savings generated as a result of the existence of a sleep center similar to the one in existence at Scripps, utilizing a capitated managed care service-delivery model.

The model was based on a capitated population base of 100,000 lives, with projections of disease prevalence based on what we believe to be the best available published data. Adjustments were made for the age distribution of patients in the served population to reflect the expected frequency of presentation of disorders. For example, relatively few newly-diagnosed cases of narcolepsy would be expected to be seen in a population with a high number of Medicare enrollees; conversely, more cases of sleep apnea would be expected to be seen in this group than in a population with greater numbers of patients in the 17-30 age range.

The existence of a sleep center would be expected to generate cost savings in several ways. The timely provision of appropriate treatments and interventions would reduce the number of office visits that would otherwise occur. For example, treating obstructive sleep apnea with nasal continuous positive airway pressure (CPAP), assuming these units are used regularly and properly, will reduce complaints generated by or exacerbated by sleep apnea, such as headache, fatigue, impotence, lethargy, insomnia, and depression. Long-term savings are also projected to occur when effective treatment is provided. Use of CPAP would reduce the likelihood that patients would later develop hypertension, heart disease, or stroke. More subtle benefits would include cost savings generated by reductions in rates of automobile and work-related and household accidents and the injuries they would have generated. The risk of developing depression may be reduced by early treatment of symptoms of insomnia, preventing progression to severe depression and the increased risk of hospitalization. Effective treatment of insomnia would reduce visits to primary care physicians for complaints of fatigue, headache, fibromyalgia, and, assuredly, for complaints of insomnia.

Projections of actual care needs for the population to be served were generated, using published estimates of the incidence and prevalence of sleep disorders, such as obstructive sleep apnea, snoring without apnea, other hypersomnias, parasomnias, and insomnia. Cost estimates were then generated by estimating the likelihood that a disorder would present in any single 1-year period. The number of sleep studies needed per year to diagnose these patients was calculated, as well as estimates of the number of full time equivalent (FTE) physicians and support staff required to care for these patients on an ongoing basis. For example, in a sleep disorders center model, it was estimated that the diagnostic, therapeutic, and administrative services needed for a population of 100,000 adults would require the services of approximately 1.5 physician FTEs.

Alternative organizational models were also considered. For example, projections were also made for the costs associated with provision of sleep care by primary physicians out-

side a sleep center. These projections included an assumption that fewer diagnoses of sleep disorders would be made and less treatment would be provided as a result of the nonspecialist approach, but that long-term costs would increase as a result of diagnoses not made and treatment not provided. This hypothesis is well supported by research in other specialty areas that has demonstrated that care provided by 'expensive' specialists leads to overall cost savings and improvement in patient outcomes.[8,9]

The costs of provision of these services were also compared with revenue allocation formulas in use at the Scripps Clinic at the time the study was performed. Comparisons were made between the actual allocated revenues for provision of sleep services allowed within this model and the projected expenditures required to care for patients in the capitated system.

Using this model, estimated savings related to sleep apnea for this theoretical plan serving a population of 100,000 would exceed $500,000/year through prevention of myocardial infarction, stroke, hypertension, and accidents. Potential savings from treatment of insomnia for a plan of this size were estimated to be $275,000. Disregarding any other factors, such as improvements in patient quality of life and in patient satisfaction, savings from prevention of these two conditions alone (sleep apnea and insomnia) more than offset the costs of diagnosis and treatment for all sleep disorders, which were estimated at $690,000.

Although the model demonstrated cost savings generated by sleep center operations, a decision to close the Scripps Sleep Center was implemented by the Scripps Clinic Medical Group administration in May of 1996. The expressed rationales for this decision were a perceived gap between allocated revenues and projected costs associated with the Center, as well as a policy statement that sleep disorders medicine was not felt to be an essential part of the core business activities of a clinic in which managed care would be the primary service-delivery model to be pursued. The closing occurred despite the implementation of many cost-saving measures; there was no opportunity provided to question whether the share of revenues allocated to the Sleep Disorders Center for its diagnostic and therapeutic activities were appropriate or what benefits and cost savings would be generated by the continued existence of the Center.

Does Sleep Medicine Save Money?

The supposition that treatment of sleep disorders may lead to long-term cost savings has been supported by several recent publications. A study by Peker et al[10] demonstrated a dramatic reduction in days of hospitalization secondary to cardiovascular and pulmonary disease in patients with obstructive sleep apnea who received treatment with nasal CPAP, compared to a period of equal duration prior to initiation of CPAP treatment. In a population of 58 subjects who regularly used CPAP, total days hospitalized per year were 27, compared to a total of 207 for the period prior to initiation of CPAP ($p < 0.0001$), with concomitant reductions in costs for hospitalization. In a comparison group, which included patients who were intolerant of or noncompliant with CPAP use, days of hospitalization and hospitalization costs were increased.

Kryger and colleagues[11] compared the health care utilization of 97 obese patients with obstructive sleep apnea and 97 matched control subjects, using data for their laboratory in Manitoba, Canada, which also served residents of Saskatchewan, Ontario, and the Northwest Territories. Over a 2-year period ending 2 years prior to the diagnosis of obstructive sleep apnea, the sleep apnea patients had 251 nights in hospital, compared to 90 nights for the control group, and were far more likely than controls to require hospitalization ($p = 0.015$). For the same 2-year period, total expenditures for physician claims for the apnea group were twice those for the control group ($p < 0.01$). When the data were analyzed over a 3-year period prior to diagnosis, the gap between apnea patients and controls was even greater.

Kryger has carried the analysis of these patients forward following initiation of treatment, looking at expenses for patients who are compliant with treatment in comparison with those who are not compliant. Patients who were not compliant did not show reductions in health care costs; the individuals who were compliant had levels of expenditure that not only were significantly reduced relative to costs prior to diagnosis but actually approached levels of the control.*

Is the Sleep Center an 'Endangered Species?'

It is clear that the existence of the classically modeled, hospital-based sleep disorders center may be endangered in environments in which managed care plays a predominant role. Many considerations may play a role in this situation, not the least of which is skepticism that this is a 'needed' service for which funds should be allocated (funds that would otherwise accrue to the bottom line, generating bonuses for physicians and administrators). This is particularly true if alternate models can be proposed that would generate cost-savings: outsourcing or contracting for services, emphasizing limited or ambulatory studies.

In addition, the sleep center within an institution may be at risk for closing if those who make decisions regarding allocation of resources see these activities as irrelevant or peripheral. This risk is reduced if the physicians involved with the sleep center hold positions of power within their institutions or if they happen to be busy practitioners who frequently admit and care for fee-for-service inpatients in their institutions.

*M Kryger, Professor of Medicine, University of Manitoba, Manitoba, Canada, 1997: personal communication.

Why isn't the diagnosis and treatment of sleep apnea perceived to be of greater value to medical systems and institutions? One reason is limitations in research data available to date, which I address later. Another is that, despite the dramatic benefits that patients experience with treatment of their sleep disorders, strong patient advocacy groups for sleep disorders medicine do not exist. There is thus no potent body to fight with Medicare or in Congress to ensure necessary services are available for the general public or for lobbying to support increases in funding for research in sleep disorders medicine. If the importance and relevance of sleep disorders medicine is not appreciated by those empowered to make the decisions regarding payment for services and the types of services to be provided, sleep disorders medicine may well be in trouble.

A problem specific to the managed care environment is the question How quick is the payoff on investment? If the benefits of diagnosing or treating sleep-disordered breathing are clear and apparent but accrue only over a number of years due to reduction in risks associated with hypertension, heart disease, or stroke, is it a rational expenditure for the 'plan' to spend dollars on sleep apnea this year, depleting the bottom line? A number of factors would make this expenditure irrelevant: the patient may move out of the area, may leave the plan, or may die from an accident or other unrelated causes. As cynical as this may seem from a medical perspective, from a straightforward economic standpoint this may make good business sense, and the death or departure from the plan of a patient may be considered a 'good' outcome.

Challenges to Sleep Disorders Medicine

Establishing the Benefits

A major challenge to the field of sleep disorders medicine is the lack of adequate research data to support a supposition that money should be spent for the diagnosis and treatment of sleep disorders. Several studies have provided data supporting the contention that there are associations between sleep apnea and disorders including pulmonary hypertension, myocardial infarction, and stroke,[12] and have shown increased morbidity and mortality due to cardiovascular and cerebrovascular disease.[13,14] Criticism has been raised, however, that these findings are based on inadequately controlled, retrospective studies.

A recent meta-analytic study by Wright[15] in the *British Medical Journal* suggests that evidence of ill effects occurring as a result of even severe sleep apnea is "conflicting and inconclusive." The title of the accompanying editorial, "A Wake Up Call for Sleep-Disordered Breathing," captures in a phrase the dilemma facing the field of sleep disorders medicine: generate data that support the contention that there are benefits associated with the treatment of sleep apnea beyond demonstrated improvements in daytime sleepiness or risk loss of support for diagnostic and therapeutic interventions for these patients.

Alternate-Site Sleep Testing

There is no question that recording at sites outside the traditional sleep laboratory is practical and can generate data that may be used to establish diagnoses and define treatment approaches for patients with sleep-disordered breathing. Such studies can be accomplished in various ways that may reduce costs by limiting the channels and physiologic parameters recorded and through unattended monitoring.

In discussing the role of testing outside the sleep laboratory, it is possible to focus too much on content and not enough on process. Our primary issue of concern should not be at what site the data are collected, but rather the types of data collected, their interpretability, and their relevance to the clinical questions being considered. Similarly, the critical question to define the quality or appropriateness of a sleep study may not be Is the study attended? The critical questions may be Are the appropriate channels selected? What is their sampling rate? What is the quality of data recorded? What is the failure rate for channels of data collected? What additional data are available to include in the interpretation? Who is interpreting the data? How are the data to be used?

Similarly, with regard to treatment initiation, it is clear that new technology has provided us with the opportunity to initiate treatment for sleep-disordered breathing in settings other than that of the most traditional hospital sleep laboratory. As with the issues relating to the study or diagnosis of these disorders, the critical question should be What allows the most effective treatment for patients and at the same time generates the best compliance with this therapy? The setting in which this is best accomplished will vary from patient to patient, and the approach to the patient must be individualized in order to lead to the best outcome.

Finally, the fact that technology exists for 'out-of-lab' diagnosis and therapy of sleep-disordered breathing does not mean that this is the best or most efficient use of technology or resources. What are limiting factors for the use of this technology in alternate settings? The home testing setting varies from individual to individual. The impact of behaviors of bed partners, children, and pets; the effects of environmental factors such as light, noise, temperature; and the intrusions generated by use of a telephone, television, or radio in the bedroom are difficult if not impossible to control in the home environment, especially if the study is performed in an unattended manner.

Despite the fact that there are credible data that automated systems, in the hands of well-trained and careful investigators, can provide data that are similar to or, perhaps, even superior to that generated by technologist-directed titration, no convincing data or other source of assurance exists to show that this technology can be effectively used in the home setting. We know nothing about the reliability of this equipment

used in this fashion or of the actual costs associated with delivery, placement, and return of the equipment, of data collection and analysis or of the necessity for repeat studies due to technical inadequacies.

A further limitation to the utility of the unattended or automated mode for CPAP initiation is compliance. Various problems are seen in the first night of use of nasal CPAP in the sleep laboratory. These include poor mask fit, intolerance of the mask or of the headgear, mask leak with movement, complaints of claustrophobia, sinus complaints or rhinorrhea, or general but nonspecific complaints—"I can't sleep with that thing blowing air up my nose." In the attended setting and even more specifically in the sleep laboratory, these problems can be much more readily addressed than in the home and other alternative settings.

In Conclusion

A sea change has occurred in many areas of the United States that have experienced heavy penetration of managed care. The changes seen include closing of sleep centers and a move to private settings rather than institution-based centers. Changes have also occurred in the types of studies performed, with greater emphasis on limited studies (including oximetry) for screening purposes, with split-night studies becoming the standard diagnostic approach for patients with suspected sleep-disordered breathing. In some settings, use of empiric CPAP in patients presumed to have a very high probability of sleep apnea has become the norm.

Patients with sleep-related breathing problems will require care, and it is clear that the health care delivery system will have to resolve the question of how best to provide this care. It can not be assured that any current delivery model based on the classic model of the sleep disorders center will (or should) continue to play a role in this process. If sleep disorders medicine is to survive as a field of medical practice, it must better understand the forces that determine what care will be given and how it will be provided, adapting to them but also trying to influence them. We must also work vigorously to obtain research data so badly needed by our field and must work with our patients to try to ensure that appropriate diagnostic and therapeutic resources remain available to care for patients with sleep-disordered breathing.

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Discussion

Barnes: I support being proactive in dealing with hospital administration. We had a hospital administrator from Pennsylvania come to our Annual Meeting to share with us his thinking. Toward the end of his presentation, as an aside, he said "Of course, you realize that I get a bonus based on how much money I save the hospital system." It appeared from the way he said it that even though he laid out these elaborate plans on why cuts should be made, self-interest was represented in his final comment. If you look at the salaries that managed care people are making, they are outrageous. He said that he welcomed physicians and technologists who were proactive and would approach him with ways to run their particular centers and departments in a cost-effective way. Physicians should be very careful about how much support they provide to technologists. Some hospital administrators try to find the cheapest way to provide technologists for sleep centers. Frequently, they

bring people off the street and train them in 6 months to meet the need. On-the-job training of technologists is not appropriate to prepare for decisions to triage your patients and direct the more severely ill. As I spoke with some of the technologists last night, it struck me that the salary structure for sleep technologists is not really what it should be. You're not going to get the people who can support you in your research initiatives unless they are adequately educated and financially compensated. I make these comments not knowing how involved many of you are with the sleep technologist organizations, with their credentialing board, with their schools. But whatever you can do to support the profession will pay dividends in the future.

Mathews: I would like to support what Tom (Barnes) said and also add that as he was speaking I saw a strong parallel between what seems to be happening with managed care—especially the example you gave with Scripps and your lab there—and what happened about 2 years ago with the drive-through delivery system—a woman having a baby and bringing the baby home within 24 hours. A massive response by the professionals involved directly to the federal and state legislatures resulted in reversing those decisions. The *Kansas City Star* published a viewpoint article by one of the vice presidents of Blue Cross-Blue Shield. She was talking about how all this legislative activity was counterproductive because, "After all, we don't want legislators making medical decisions." I got a little 'ripped off' and wrote a responding letter, published in about 20 newspapers and several journals, that pointed out that we would far prefer a legislator who had no financial stake in this to make those decisions rather than to have insurance people make them.[1] We must realize that we set up this situation. We allowed the insurers to make these decisions, and we're letting them get away with it. But maybe that is the 'blue cross' we all have to bear. We got ourselves there, and we've got to carry that unless we can change it.

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Estes: Milt, I liked your model, looking at costs. One thing that struck me is that you were trying to build it from the ground up. Kryger[1] recently looked at data from the Canadian health system that might be helpful to someone else having to build a model. Untreated OSA patients incurred $200,000 more in a 2-year period than when they were treated—and that's for 97 patients. It works out to about $1,000/year, which is almost the premium that you were talking about. The second thing, in your model the rate of effectiveness was about 50%. If there's any argument that tells us we should be worried about compliance and need to look at who is compliant and who is not and work on making the noncompliant patient more compliant, that 50% effectiveness is the most telling number of all.

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Erman: Absolutely. We believed, of course, as does everybody in the room, that our compliance rates were really much better than that, but we thought we had to stick with the published data. But if we really believe that these cost savings exist, then, if we improve compliance, there's a multiplier effect. By doubling compliance, which is probably impossible, or by a 50% increase, you have very substantial cost savings with marginally increased costs.

Chediak: One of the themes that I have heard yesterday and today is our lack of data to support our interventional outcomes in patients with sleep disorders, both sleep apnea and others. I wonder, realizing how long it takes to get data collected and published in the mainstream, through the NIH and other funded organizations, do you advocate that sleep disorder centers collect data as best we can on our own populations and develop our own models so that we can, if need be in the future, present that information to administrative and capitated sources of managed care to document our contributions to patient care—supporting our existence in that manner?

Erman: Potentially the data that may be valuable in those contexts within institutions may relate to what can be documented in terms of cost-savings. Data not yet collected or presented would be valuable—things like demonstrating what cost-savings are, as Kryger has done, to show that if you treat these patients hospitalizations go down; the cost of care goes down. In the larger sense, what we need are the types of studies that were criticized in the Wright article[1] for not existing. We need studies that are well designed, that are prospective. Some are ongoing now, such as the Sleep Heart Health Study. More of these are needed to provide the data that I think that we all believe should exist. Virtually everybody in the field and everybody who has worked in the field believe that there are costs associated with these disorders. We may believe that the treatment reduces the risks and costs, but the problem has been the lack of data that are not polluted by factors like obesity, age, and other illnesses, which allow questions to be raised about the validity of the associations.

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Strollo: There are two issues here with the transition to managed care. One is the ability to deliver care to a needy population and to continue to exist to be able to do that. The other is improving knowl-

edge in the area of how we deliver care, ie, research. That's probably one of the great concerns with the model for managed care—that research is not built in. You've thought about this a lot. How do you see our system being able to dovetail the component of research?

Erman: Scripps is a curious animal because it is not a classic medical center, and it doesn't have a large endowment, the way a place like Mayo has. So, you have to look at it as being somewhat anomalous. I was unfortunate to have been the head of the center of the anomaly, but that's the reality. It is going to be increasingly difficult in many of these settings to do things like research and fellowship training. These will need to be provided by academic medical centers with access to traditional sources of funding. We are continuing in our new private setting to do research, some with federal funding through Merrill Mitler's grants and some with industry funding. It's possible to 'piggy back' research onto some other sources of funding, but it is quite difficult. I should add that the system has not totally done away with care for patients. We, in fact, at our new center at Pacific Sleep Medicine Sources, provide the care on a contracted basis to the clinic at a cost that is certainly lower than it was in the institution. So, from a rational business standpoint, it can be argued that this was a good business decision. The problem is, of course, what is lost to the institution in terms of the existence of the center onsite—for example, the research training and the collaborative relationships. We can understand that there are ways of providing care in more cost-effective fashions, and we certainly need to be driven in that direction, but the future is likely to include changes of this sort in other settings and at other institutions, as people look at the bottom line and simply say "If this operation is closed, who will notice, what will the consequences be, and what are the cost savings?"

Strollo: I have one more question and direct it to industry. We're having increasing pressures to partner with industry with regard to research, and, frequently, that's the solution, but industry is under the same pressures that other health care delivery systems are under, and there's a fixed budget. How do you guys see industry dealing with the increased burden of assuming the cost of research?

Estes: On the research side, we are having to spend a significantly higher percentage of sales to even keep the same level of research going. It's pretty hard. We really are focusing on research that will improve the outcomes that will keep our industry alive. So, if you look at the research that we're looking for and supporting, it tries to prove that things would help—convince managed care to treat sleep apnea, for instance, or convince managed care that home mechanical ventilation is a good thing. Those are the kinds of things we're after. The other thing we're doing, independent of research, is expending a lot of effort trying to put educational tools together that can reach the managed care person who's making that decision based on numbers, trying to put believable tools in their hands. Those are hard to come by. So, that's a lot of effort. Those are the two approaches we're taking.

Atwood: Will you comment on how the sleep physician should partner with the primary care physician? You presented econometric data about turning some of the work that you or sleep physicians do to primary care, but you didn't really define exactly what that level of responsibility for the primary care physician would be. How do you view your interactions with them now? Can we teach them to do what we do to some extent, save ourselves for the more complicated patients, and try to carve out an even more defined or better defined niche for ourselves that may end up protecting us in the long run?

Erman: I think to some degree we can give some responsibility to the primary care physician. The epidemiologic data suggest that huge numbers of patients have not yet been identified. The problem really is how do you provide the care? How do you identify patients? I think we can do a better job of educating primary care physicians to recognize the symptoms that should lead to consideration of sleep disorders. Even at Scripps where we had a sleep center in existence from 1982 through 1996, 14 years, there was a tremendous amount of variability within the primary care population with regard to the knowledge base, the sensitivity, and the referral rates. But if the primary care people recognize that these disorders exist, that they and the patients benefit from appropriate referral, then I think it can be done. A model that may work is based on putting limited power in the hands of the primary care physician. It is not really an efficient use of resources to have people making empiric decisions, saying "Well, I think this is a patient who should be on CPAP. His wife tells me he just snores up a storm and he tells me he's tired."—without knowing that that patient has sleep apnea. The cost of the study is probably rational in terms of being able to determine what the treatment options are. Also I think that the added cost of professional consultation in a capitated system is minimal. It really doesn't cost much more to have that patient sitting in *my* office as opposed to the primary care physician's office for the 30 or 45 minutes of the evaluation. We must work in a smarter fashion, doing things like using oximetries. We know the limitations of oximetries, but an oximetry, combined with an Epworth, combined with an evaluation by a practitioner who is knowledgeable, does give some power and helps to focus utilization of resources in ways that can be more cost-effective. We may encourage screening by the primary care people. Another model may say, "We'll just let the primary people take care of them." The assumption is that it costs less because you don't have a lab, but the actual cost to the system may be more because you don't have the cost-savings associated with case findings and treatment.

Minkley: I think Ball and Simon[1] in the Walla Walla project described the involvement of primary care fairly well in working in a clinic where there were no sleep diagnoses and educating the clinicians and seeing, of course, an increase. In addition, at least one of those physicians involved ultimately became a sleep specialist. Duplication of the Walla Walla project has been tried in other areas, but the effort actually hasn't been really successful, primarily because no clinician wanted to embrace sleep disorders and actually take the lead and learn sleep disorders to the depth that is necessary to actually manage the patients. That's a big concern.

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Erman: Yes. Otherwise, you're simply encouraging the primary care people to take on additional responsibilities that they don't feel terribly comfortable with. This takes more time and doesn't provide them with resources to adequately take care of the patients. That's not a very fair tradeoff.

Minkley: That is the place where in some ways, as was described a little earlier, that if there is an existing sleep center, if there are individuals trained in the technological aspect, there can be some direction if, at least, those patients come to some central clearinghouse. But the challenge seems to be that at the same time we need this central clearinghouse or a place where the knowledge base is broad in sleep disorders medicine. The picture and the structure of sleep centers is changing to become more fragmented, just as you've described where it's privatized. Where there's a diagnostic center or physical space, the physicians and the clinicians who are skilled in the full range of sleep disorders are in separate private practices and may not be in a group as you described, but, literally, geographically spread across the city. It becomes very difficult to coordinate these aspects of care.

Stoller: I have a question that deals with the philosophy of data. I'm sure you've thought about this. You have challenged us to generate data about the value added, for example, of a sleep disorder practice and practitioners, and the AARC faces the same challenges with regard to respiratory care overall. In that regard, the sleep-disordered breathing community, the AARC, and many others have gone about gathering data to critically examine the "value added," of their respective expertise. The question naturally arises: Are such data—coming from a group whose purpose is served by demonstrating their value added—tainted? In other words, could they be good data, but disregarded just as we heard Tom Barnes describe data coming from managed care organizations, where the administrators have the incentive to reduce costs? Are the data tainted in the perception of the recipients of the data? So, the generic question for the sleep-disordered breathing issue and for respiratory care is: Does the source of this economic data or value-added data need to be a source other than the groups directly involved in the ascertainment of benefit? Does NIH, does industry, does someone who is an impartial, dispassionate health care resource group need to engage the issue of gathering data, or can we do it in a meaningful way? It's obviously an unanswerable question, but I think an important one that impacts on a lot of activity that's going on right now.

Erman: I think it is answerable, and I think the point is well taken. My exhortation is more to the field in general. We need good science. We need scientifically valid studies, prospective studies, studies that are based on good epidemiology. This is based on a belief system that I think is shared by most people who work in sleep-disorders medicine, including a belief that there are health consequences of these disorders and that their treatment generates health benefits, cost reductions, and clear cut improvement in quality of life. Now, we can't go out and simply preach based on our belief system. What I'm saying is that I'm hopeful that these data will be available in the near enough future that they can provide us the opportunity to continue appropriate work in diagnostics and therapeutics. This is absolutely dependent on having appropriate scientific research, well-validated, well-designed, and capable of standing up to rigorous scrutiny.

Strohl: The major role that successfully-treated patients can play is as advocates. The American Sleep Apnea Association (ASAA) is a group oriented toward sleep apnea patients that's been supported by respiratory therapy and sleep technologists. It was developed by Lucy Segar. The ASAA hasn't reached a critical mass yet, but it's been in existence only since 1991, and I would encourage membership by physicians, respiratory therapists, and sleep technologists. It's not just for patients. The second is the National Sleep Foundation, which is an umbrella organization for all sleep disorders. Have you been able to engage patients and others in this way in California?

Erman: Yes and no. We have an A.W.A.K.E. group, for example, that has been quite active. Advocacy groups of this type can certainly be used to try to influence policy and funding. If we simply look at the allocation of resources at a national level for various diseases, we see that they're not based on prevalence or survival data. They're based on political issues of what disease is 'popular,' what is perceived to be the appropriate expenditure. Heart disease, which on a rational basis should have higher funding than cancer, certainly doesn't, and certain 'popular' diseases have grossly disproportionate funding relative to their incidence rates and severity of illness in the population. I think the role of the patient groups can be to try to influence health care policy and

research support, to benefit the field, and hopefully benefit society in general.

Strohl: We have tried a couple of things in Cleveland that are interesting. One was a 'sleep-in' of patients with sleep apnea, in 1991. They came to a local hotel, paid for a suite of rooms, and encouraged media attention as well as an opportunity to do things. New portable monitor technologies from the different companies were tried out in patients who knew what it was all about. A current idea is to have officers in the A.W.A.K.E. group to create a "Mobil" guide to sleep labs in the community. The best sleep lab would be the CEO's office at night. It would be attractive and cost-effective.

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Respiratory Care Practitioners and Sleep Medicine: Opportunities and Challenges

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Background & Introduction

Sleep disorders medicine is a new field. Clinical sleep studies were still a rare inclusion in medical diagnostics in 1972 when Guilleminault came to the Stanford University Sleep Center and began adding respiratory and cardiac sensors to sleep studies. In 1974, Holland (also at the Stanford University Sleep Center) named the new generation of sleep studies polysomnography, but it was not until 1976 that daytime and nighttime sleepiness were recognized as a continuum. The U. S. medical community began in earnest to observe the sleeping clinical patient, and polysomnographic technology began to be recognized as an allied health career option. The Association of Polysomnographic Technologists (APT) had formed 7 years earlier, in 1969, with a handful of members, and now has a membership of nearly 2,000. Most of the growth has occurred since the middle to late 1980s. Sleep medicine technology, encompassing polysomnography, has grown into a highly specialized and complex allied health field. Opportunities in sleep medicine technology are exhibiting a steep growth curve largely due to the public education efforts of the National Commission on Sleep Disorders Research that has alerted the nation to the hazards of sleepiness. At present, many patients seen in clinical sleep centers come with an admitting diagnosis of obstructive sleep apnea (OSA) and are frequently initially treated with nasal continuous positive airway pressure (CPAP). Although the terms apnea and CPAP are familiar to the respiratory care practitioner (RCP), their application in clinical sleep medicine differs from their application in the intubated acute care patient. RCPs have a strong base upon which to build a career in polysomnography. However, additional education and training are essential for the RCP to function safely and effectively in sleep medicine—to appreciate distinctions among the 77 identified sleep disorders and to effectively and appropriately apply noninvasive CPAP and bi-level ventilation in the setting of a clinical sleep center or any of the other venues addressing the care of the patient with sleep-disordered breathing.

All of us spend a significant portion of our life asleep. We take this portion of our life largely for granted, and as a society voluntarily decrease the one third of each day we should spend sleeping to levels that sometime affect our ability to function and learn—without even recognizing the effects of our choice. We may believe that we have intimate familiarity with our own and family members' sleep. This familiarity challenges the sleep medicine and sleep technology community to dispel the underestimation of the complexity of skill

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required to study a sleeping patient. Treatment-intervention protocols based on the observed pathologic events and physiologic responses of the sleeping patient are added to complex observational skills. I believe that functioning safely, efficiently, and effectively as a polysomnographic technologist demands specialized education and training.

In this paper, I focus on the complexity of skills required to function as a polysomnographic technologist, looking at the past, present, and future growth in the field of sleep medicine and technology, the opportunities being created, and the preparedness of the practicing and new graduate RCP to enter this growing allied health field.

Publications related specifically to competencies required by polysomnographic technology are limited. I gathered statistics on the growth of sleep medicine and polysomnographic technology from both literature searches and professional organizations associated with sleep disorders medicine and technology. Information was obtained through discussion with colleagues performing polysomnography or associated skills in traditional sleep centers and alternative settings, such as home health care, acute care, physician offices, and nursing facilities. Educators in sleep medicine and technology contributed information on the background of those seeking training in the field, and manufacturers' representatives provided a view of the environments in which sleep technology is being sought and sold. Abbreviations and a glossary of terms appear in Table 1.

History of Sleep Medicine & Technology

Rapid eye movement sleep (REM) was identified in 1953,[1] increasing interest in the study of sleep. A clinical sleep center was opened 7 years later at Stanford University, with the focus primarily on narcolepsy, and sleep studies rarely recorded

Table 1. A Glossary of Terms Used in Sleep Medicine

Association of Polysomnographic Technologists (APT)	a professional organization for persons active in polysomnographic technology, primarily polysomnographic technologists.
American Sleep Disorders Association (ASDA)	an organization for accredited sleep centers and laboratories and individuals active in sleep disorders medicine and/or polysomnography, primarily MDs, DOs, PhDs, and DDSs; formerly Association of Sleep Disorder Centers (ASDC).
Bi-level ventilation	noninvasive positive airway pressure with independently set inspiratory and expiratory pressures.
Board of Registered Polysomnographic Technologists (BRPT)	credentialing board, responsible for administering and maintaining the Registry examination process and Registry membership for polysomnographic technologists.
Continuous positive airway pressure (CPAP)	may be administered noninvasively by nasal or full-face mask when used therapeutically in association with sleep-disordered breathing as opposed to the invasive use in intubated acute care patients.
Electroencephalography (EEG)	a recording of the brain's electrical activity.
Electroneurodiagnostic (END)	an allied health field that includes EEGs, evoked potentials, nerve conduction studies, and other neurodiagnostic testing procedures.
International Classification of Sleep Disorders (ICSD)	a diagnostic classification and coding manual with standardized descriptions of sleep-related abnormalities, published by the American Sleep Disorders Association in 1990; revised in 1997.
Multiple Sleep Latency Test (MSLT)	an objective measure of a subject's ability to fall asleep at multiple nap opportunities the presence or absence of sleep-onset rapid eye movements; an objective measure of sleepiness and the possibility of narcolepsy.
National Commission on Sleep Disorders Research (NCSDR)	commission appointed by Congress whose September 1992 report to Congress and the Department of Health & Human Services resulted in the formation of the National Center for Sleep Disorders Research of the National Institutes of Health.
Nasal CPAP (NCPAP)	nasal continuous positive airway pressure; term used to differentiate invasive and noninvasive applications of CPAP; see CPAP.
Obstructive sleep apnea (OSA)	sleep apnea characterized by soft tissue obstruction of the upper airway (as opposed to sleep apnea stemming from abnormal respiratory control).
Polysomnogram (PSG)	a multiple-channel recording of physiologic and behavioral variables occurring during > 6 hours of sleep.
Respiratory care practitioner (RCP)	a clinician licensed and/or credentialed to provide diagnostic, therapeutic, and life-support services to patients.
Registered Polysomnographic Technologist (RPSGT)	a credentialed clinician who provides diagnostic and therapeutic services to patients with disturbances to their sleeping and waking cycles.

anything but brain waves. In 1968, Rechtschaffen and Kales published the first manual for scoring sleep stages.[2] Not much was known in the U. S. about OSA until it was described by European researchers in 1965.[3,4] Guilleminault came to Stanford in 1972 from France, bringing extensive experience with OSA. Respiratory and cardiac sensors were added for sleep studies at the Stanford clinic, and the first day-long course in diagnosis and treatment of sleep disorders was offered by the university. By 1974, human sleep was clinically evaluated by placing electrodes and sensors, indicators of brain, cardiac, and respiratory function, on patients and observing their sleep. This new generation of sleep studies was termed polysomnography by Holland, a physician at Stanford. By 1976, daytime and nighttime sleepiness were recognized as a continuum, the Multiple Sleep Latency Test (MSLT) was developed, and sleepiness began to be recognized as an important clinical symptom.[5-7]

Work done in the latter half of the 1970s established sleep, sleepiness, and OSA as legitimate clinical concerns. By the end of the 1980s, guidelines for cardiopulmonary sleep studies and the MSLT[8,9] and the first sleep medicine text book[10] were published. Guidelines and an accreditation process for sleep centers were developed by the Association for Sleep Disorder Centers and examination processes were in place for certification of physicians, doctoral-level clinicians, and technologists; and the first international classification of sleep disorders was developed and published.[11] Polysomnography guidelines were published in 1994 and 1995.[12,13] Accredited sleep centers have grown from 3 in 1977 to 337 in 1996 (Fig. 1). Accredited and nonaccredited centers may number as many as 3,000 today. The numbers of credentialed sleep specialists and technologists have risen even more dramatically.

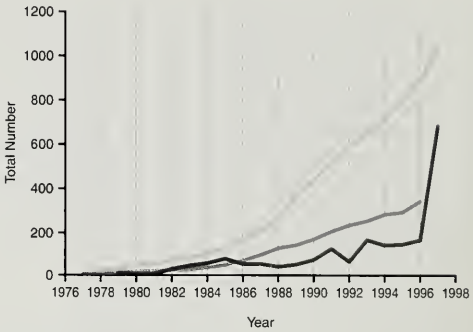

Fig. 1. Growth of Registered Polysomnographic Technologists (RPSGT), accredited sleep centers/laboratories, and credentialed sleep physicians. = RPSGTs; = accredited sleep centers/laboratories; = credentialed sleep physicians. The data was provided by the American Sleep Disorders Association and the Association of Polysomnographc Technologists.

Polysomnography Today

More Than a Sleep Study

Polysomnography borrows and adapts technology from electroneurodiagnostics, pulmonary diagnostics, respiratory care, biomedical science, and other fields. It requires adaptation of clinical aspects of neurology, pulmonary, and sleep medicine with aspects of gastrointestinal medicine, ear, nose, and throat medicine, urology, psychiatry, psychology, and social work. It is an amalgamation of skills that is a specialty unto itself—sleep medicine and technology. In the same sense that mechanical ventilation is more challenging than pushing air into a human being and allowing it to exit naturally, polysomnography is more complex than simply making recordings while the human subject is sleeping. A polysomnographic technologist requires knowledge and skill from electroneurodiagnostics for an understanding of neurophysiology, the international 10-20 system of electrode placement, recognition of brain seizure activity and pathology; for an understanding of alternating current amplifiers, filters, sensitivities, and operating principles of the polygraph; for electrode placement for electro-oculography (EOG) and electromyography (EMG); and for the ability to read electroencephalography (EEG) recordings at a paper speed of 30 mm/s. From respiratory care and pulmonary diagnostics, the polysomnographic technologist requires knowledge of pulmonary anatomy and physiology; pulmonary mechanics; operating principles of pulse oximeters, strip-chart recorders, end-tidal and transcutaneous CO_2 monitors; of principles of oxygen administration and ventilation; and of acute and emergency care. It is important to remember that the physiology studied in both electroneurodiagnostics and respiratory care rarely includes mention of variations of that physiology during sleep or during sleep onset and arousal that is crucial for the polysomnographic technologist to understand fully. The polysomnographic technologist borrows skills and knowledge from various other allied health specialties to troubleshoot equipment interfaces; to set up and monitor strain gauges, thermistors/thermocouples, esophageal pH probes, esophageal pressure monitors; and to interact with psychologically unstable and sleep-deprived patients.

Technologist Competencies

In 1989 and again in 1995, the Board of Registered Polysomnographic Technologists conducted a nationwide job survey and task analysis.[14] While the tasks already described were included, they had to be modified and adapted for use with the sleeping patient, and many of the skills included were unique to sleep medicine and technology. For example, the electroneurodiagnostic technologist is skilled at identifying recorded brain waves in the clinical EEG. To identify recorded brain waves in polysomnography, that technologist must learn to identify waveforms from referential (as opposed to bipo-

lar) EEG derivations recorded at 10 mm/s rather than 30 mm/s. In addition, polysomnography requires recording of alternating and direct current physiologic potentials unfamiliar to electroneurodiagnostic technologists, using amplifiers and filter and sensitivity settings that are also unfamiliar. Even skilled respiratory therapists are unfamiliar with many electroneurodiagnostic competencies. The polygraph is unfamiliar as are the pulmonary physiology and mechanics of the sleeping patient. Physiologic variances in cardiopulmonary function, thermoregulation, and gastrointestinal function during sleep are rarely part of an allied health care educational program.[15-17] The scoring of recordings from the sleeping subject for sleep staging and clinical events, recognition of normal physiologic variances during sleep, and the conducting of multiple sleep latency testing to assess sleepiness are unique to polysomnographic technology. The polysomnographic technologist must be familiar with known and proposed sleep pathologies to assess the appropriateness of the ordered study; anticipate behavioral activities during specific sleep stages and sleep periods; balance intervention with the need for diagnostic data gathering; and accurately identify polysomnographic and behavioral features during the study and while it is being scored. For example, a suspected sleep walker is more likely to show this activity early in the study period when Stage 3 and Stage 4 sleep are more abundant.[18] The subject with sleep apnea is at greatest risk late in the study period during the longer periods of REM sleep when vulnerability is greatest.[19] The detailed list includes more than 300 tasks.

Polysomnography Tomorrow

Opportunities in Sleep Medicine & Technology

Sleep-disordered breathing is one of the most extensively studied of the sleep pathologies and is reported to have an incidence of 2-4% of the population, whereas the incidence of narcolepsy is estimated at 0.1%, and at any given time 100% of us have experienced daytime sleepiness and insomnia. The National Commission of Sleep Disorders Research (NCSDR)[17] estimates that 40 million Americans are chronically ill with various sleep disorders while 20-30 million experience intermittent sleep-related problems. The report states "overwhelming evidence from testimony and specific surveys suggest that the vast majority of Americans with sleep disorders remain undiagnosed and untreated," they affect all age groups, and the incidence will increase. The cost of these undiagnosed sleep disorders can be measured by dollars, injuries, performance deficits, and increased utilization of health care services. Direct costs were estimated at 15.9 billion in 1990.[17] Undiagnosed parasomnias can result in personal injury. The primary symptom of undiagnosed sleep disorders, sleepiness and/or fatigue, can result in performance deficits that negatively affect job performance, decision making, and learning ability.[18-21] Medical disorders such as asthma can be negatively affected by lack of sleep,[22,23] resulting in increased use of health care resources. The high incidence of undiagnosed sleep disorders and potential for health care savings if diagnosed and treated are optimistic signs for increasing opportunities in sleep medicine and technology. Where will those opportunities be? The increase in the number of technologists and physicians seeking certification in sleep medicine and technology plus the steady increase in the number of sleep centers seeking accreditation suggest the study of human sleep is growing steadily. Because each segment of our society spends at least part of its day attempting to sleep, employment opportunities may be present in all segments. Public education about sleep disorders and professional education in the diagnosis and treatments of sleep disorders lag far behind potential growth.[17] Because all human biologic systems are affected by the quality of sleep,[23,24] treatment is frequently a multifaceted approach that requires cooperation between allied health and medical professionals, providing opportunities in case management for the skilled polysomnographic technologist. Educational opportunities exist in allied health programs, medical school programs, industry, and health care practices. The NCSDR found awareness of sleep disorders and the consequences of their remaining untreated to be absent and pervasive.[25] The sleep-center model originally included a clinical facility for initial evaluation and follow-up in the same setting with diagnostic testing. Diagnostic testing is now frequently separated physically from clinical facilities. A well-trained polysomnographic technologist can function effectively in clinical facilities as a physician extender and case manager. Other opportunities likely exist in acute care. A patient who sleeps well may respond better to treatment and be released more quickly from the expensive acute care facility. Extended care facilities may benefit from a polysomnographic technologist who can screen patients for sleep disorders; educate patients and staff about sleep hygiene, circadian rhythms, and the night-day continuum; and ultimately aid residents in attaining productive and active days and restful nights.[26,27] School systems could benefit from the same education. Opportunities may exist in industry for diagnosis and treatment of sleep disorders to produce safer workplaces and in clinical application and sales. The home care industry can benefit from broadly trained sleep medicine and technology professionals to educate all patients about sleep disorders—fatigue and sleepiness as it relates to their treatment —beyond initiating, maintaining, and following up noninvasive positive airway pressure treatment. Finally, the broadly trained sleep medicine and technology professional may contribute to and find opportunity in clinical and basic science research.

Challenges for the Respiratory Therapist

Just as all that wheezes is not asthma, all who sleep poorly are not sleep apneics. Sleep apnea often co-exists with other sleep disorders such as narcolepsy, periodic limb movements

in sleep, esophageal reflux, and poor sleep hygiene. In the same sense that a specialist in emphysema with no education in asthma, bronchitis, pneumonia, tuberculosis, sarcoidosis, or other pulmonary diseases would be ineffectual, so is a sleep technologist who sees only OSA. We must broadly define sleep study to include more than sleep apnea. Respiratory therapists must educate themselves in all aspects of sleep medicine and technology. They must work with other allied health professions to establish training programs.[28] They must learn new applications for their acute care skills in the sleep center and add sleep medicine and technologic competencies to their skill sets. Recommendation 5 of the National Commission on Allied Health[29] states "allied health professionals...[must] collaborate to foster understanding and appreciation of one another's unique and collective interests and capabilities." Recommendation 6[30] encourages allied health professionals to "... undertake efforts to reduce existing barriers to clinically effective and cost-efficient scopes of practice for those whose scope of training currently exceeds their scope of practice and for those who add new or multiple competencies in the future."

In Summary

The field of sleep medicine and sleep technology is growing exponentially. While competencies include aspects from many allied health fields, it is a unique specialty requiring a skill set not available exclusively from any one of those fields. Respiratory care and electroneurodiagnostics contribute many of the educational competencies that provide a base upon which to build. The great influx of technology challenges the field for scientific evidence of indications and contraindications of its use; techniques for its use; and outcomes of its use. This technology provides the ability to study most human biologic functions during sleep and is not limited to pulmonary diagnoses. Sleep apnea cannot be studied or diagnosed in a vacuum. Sleep disorders often co-exist and if one is left untreated, the consequences of the primary symptom, sleepiness, can be costly and devastating. Treatment interventions require broad knowledge and skill in sleep medicine and technology. The expectation of patient driven protocols is to decrease expense by appropriately matching diagnosis and treatment with patient needs. To accomplish this, respiratory therapists must gain knowledge of all 77 identified sleep disorders, the 11 proposed disorders, and the ability to recognize the co-existence of these disorders. In addition, they must learn how cardiopulmonary physiology and neurophysiology vary from wake to sleep and new applications for their technical, diagnostic, and assessment skills. Respiratory care practitioners bring excellent basic science and cardiopulmonary physiology knowledge, and technical, assessment and critical thinking skills to sleep medicine and technology, but they must gain domain-specific content and procedural knowledge to function safely and effectively as polysomnographic technologists in sleep disorders medicine and technology.

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Discussion

James: Would you comment on current legislation in several states that actually prohibits sleep technologists from performing most of those competencies that you have shown on the screen.

Minkley: In some states where respiratory care licensure exists, sleep centers are being challenged by licensure boards to assure that there are respiratory care practitioners (RCPs) on every shift because nasal CPAP or CPAP is instituted and oxygen is administered, and they are interpreting that as a scope of practice that is protected, even when the center has registered polysomnographic technologists on staff. What I really need to say here is individual states are challenging this. If you look at all the presentations we've had here, I think you can appreciate that fresh out of school, a respiratory care practitioner probably doesn't have the competencies required to function adequately in a sleep center. They have studied CPAP and oxygen administration, but the application of these modalities noninvasively to the sleeping patient and other sleep-physiology-based competencies are rarely part of the curriculum. The Model Practice Act for Respiratory Licensure does actually have a clause in it that says that the licensure is not intended to be exclusionary, but if other allied health professions have demonstrated competencies in an area, they may be included and be able to practice. The registered polysomnographic technologist (RPSGT) credential specifies demonstration of competency. The respiratory care licensure being invoked by mandating the presence of respiratory care practitioners in sleep disorder centers, solely because they hold that credential and licensure, may not have that clause. This is of concern to a great many people. If the ultimate goal of licensure is to protect the safety of patients who seek care, are we, in fact, doing that if we put practitioners in the position to do studies and to actually interact with patients without the knowledge base that makes them safe and able to practice? This is not to say RCPs should not be or do not make good sleep medicine and technology practitioners. But holding a respiratory care credential, in and of itself, likely does not at this time provide the sleep medicine and technology knowledge base and competencies necessary. So there's a great deal of concern right now about those issues.

Brougher: You pointed out the Clinical Practice Guidelines and particularly the one on polysomnography[1] that takes the position of being totally non-turf-protective and emphasizes the importance that the person working in whatever capacity in sleep medicine has a documented ability and knowledge to perform the tasks. I think I can speak for the Association in this regard—that is more nearly the position of the Association than any arbitrary turf-protecting approach.

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Mathews: I can testify to that, as I am one of the people who wrote the Model Practice Act[1-3] a few years ago, and that was the intent.

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Minkley: The Clinical Practice Guidelines?

Mathews: No, the Model Practice Act. The intent was that if people were trained in that area, we weren't going to restrict them for one or two little bits of the scope of practice.

Minkley: I think you also demonstrated that when you included the cardiopulmonary technologists and accepted some of their skills and practices and then developed the pulmonary function credential and diagnostic sections.

Mathews: A question for everybody: Do you think that the general public thinks that snoring is a normal phenomenon? Do you think that they do not see this as abnormal?

Minkley: Oh, sure. That is likely the case for many clinicians also.

Mathews: So this is a PR [public relations] problem. Is that correct?

Minkley: Sure. The National Commission on Sleep Disorders research pointed that out clearly, and I'm sure the clinicians here can probably attest to that, also. There's some disagreement about the severity of the impact of snoring. We talked about it, too, when the issue was brought up of nonpathologic soft snoring. Is it still an issue to be dealt with, if it drives the bed partner from the bedroom? I don't think as a medical community we yet truly look at the social aspects of any disorder, but the system is still based on finding something that's broken and fixing it. Social aspects or psychological aspects usually aren't considered as part of that 'broken' aspect.

Mathews: The second point—in one of your slides you talked about transient sleep problems that everybody has. Does that appear to diminish, in people's minds, the importance of sleep disorders? Because "We all have them! I 'catch up on my sleep' and I feel fine."

Minkley: I think that is true. We all feel very intimately associated with sleep. Just take the evolution that's going on right now in the training of interns and residents. The sense for a long time was, "Look, I made it through my residency. I did 4 days without any sleep. By George, that's the price that people pay, and it's a badge of courage." Now we actually look at the incidence of errors as people are sleep-deprived and say "Gee, maybe we really should look at that." Look at napping. We know with narcoleptics and for some people with hypersomnolence that napping is very restorative—but, yet, as a society, if we see a person taking a nap, we don't look at them in the same way as the person who runs by in his jogging shorts. About the person in jogging shorts, we say, "Wow! Look at him. He's taking care of himself. He gets regular exercise. He's jogging." Yet maybe the person taking a nap is, in effect, doing the same thing, but we see that person as being lazy. Or if they're sleepy, then they aren't in good control of their own being because if they're sleepy, they should just get more sleep, and they don't.

Mathews: One of the things that we've noticed since we've started teaching our students more and more about sleep is that they're becoming gatekeepers. They walk through the hospital corridors at 1:00 AM, 2:00 AM, they hear this tremendous snoring, then absolute silence, then a snorting sound—that big jump when the patient wakes up. They go down and talk to nurses and say, "You need to have somebody look at this patient and look for sleep disorders." So, I think there's a real role for the RCPs to serve as kinds of gatekeepers for patients who may not have been picked up by their physicians or surgeons.

Minkley: Broadening the spectrum of sleep education for respiratory care has another aspect. Dr Mary Carskadon has done a lot of work looking at sleepiness and our inability to recognize that students are one of the most underslept populations. Look at our respiratory care students who frequently combine work and respiratory care education, and, being the 'new kids on the block,' when is most of their work? Weekends and midnights, and then they squeeze in school. We now recognize that both long- and short-term memory are affected by lack of sleep, so here are students who have behaviors that they don't know about that may be compromising their ability to do what they came there to do—learn! In addition, we may be able to help these people recognize other issues. If you walk through the hospital at night, you see people at nursing stations with blankets over their back at 3 & 4 AM and that's because that's the time their normal circadian dip occurs, where they have the most difficulty staying awake. One of the things that increases wakefulness is activity, so we can help respiratory care practitioners to understand sleep hygiene and circadian rhythms and to say that's not the time you want to sit down and do something quiet in a dark corner like chart or check med cards because you're likely to make errors. Do something like checking the stockroom, going down and working in the parts room and cleaning ventilators—something that has a high level of physical activity that's stimulating. There's a broad range of knowledge that we haven't even tapped and that respiratory care practitioners can look at by being trained in the full spectrum of sleep disorders and sleep itself.

Barnes: We can cross-train respiratory therapists to work in sleep centers or we can teach them more about sleep disorders. I'm sure we could teach them more about sleep disorders. I'm unsure whether we should train them to work in sleep centers. My experience with perfusion technology has been that when we trained people to become perfusion technologists, they left respiratory care

and became part of the cardiac surgery team. In other words, they were no longer respiratory therapists. They were perfusion technologists. Quite frankly, there's not much in it for respiratory care educators to take their best students and train them to work in sleep centers. I think there's a lot in it for us to train them about sleep disorders, and I certainly will do that, but I would feel better in terms of the cost-effectiveness of health care delivery if that someday we could educate perfusion technologists and sleep technologists so that they could work in all of these areas and not pigeonhole them on a heart-lung machine or put them in a sleep center and say that that's all they're going to do, especially for a sleep center where the patient load may vary. It could be that a person could work on the 4th or 5th day of the week as a respiratory therapist and the other 3 days of the week in a sleep center. This question needs to be answered because it's going to affect what you do as a sleep technologist organization. Are you going to decide, for instance, to become a membership section of the AARC and approach the National Board for Respiratory Care to develop a sleep technology specialty exam for respiratory therapists?

Minkley: The registry exam in polysomnographic technology was developed under the same guidelines and by Applied Measurement Professionals (AMP), the same group who develops the respiratory care examinations. But, I think we're forgetting our friends and colleagues from the EEG side. In the spirit of the Allied Health Commission, I think we do need to work together and do just what you're talking about—not educating people in pigeonholes, but broadly educating them. If one of the costs associated with sleep centers that we didn't look at when Dr Erman was speaking stems from the lack of schools to train these people (and I think that you brought this up, too), the cost of training because they have to be on-the-job trained, whether they come out of EEG or respiratory care or off the street. There's a new application of the skills they already have and then additional skills and competencies that need to be learned. The cost of this training is tremendous on the job. If we don't treat well-trained people well, turnover is constant, which adds costs to the system. From a strictly financial standpoint looking at base salaries, if you don't integrate those turnover costs, it may actually cost more to pay people less and give them fewer benefits than it does to pay them more initially, but keep them longer. Because competencies do overlap (EEG, respiratory care practitioners, and so on) there would be some cost savings to the system to train them as a group. The same thing occurs with nursing procedures, patient assessment, all these issues that overlap. If we could train these people together, certainly we could produce practitioners who are better based in basic sciences as well as technology, and then they could add specific competencies on top of that.

Barnes: It seems to me the most valuable sleep technologist is one who can function as a physician's assistant (PA). The National Academy of Science and Science Medical Board, about 20 years ago, identified a specialist-type physician's assistant as one with more knowledge in the area of specialization than the average physician has. That's not to say they would have more knowledge than a physician specializing in sleep medicine. However, the PA would have more knowledge than the average physician—a valuable person to put into a sleep center because he/she could do the research needed to help make the field of sleep technology progress.

Minkley: You are right. Realistically, though, the only way to get reimbursement would be to train as a PA, get credentialled, and then come back and find training. I don't know how to deal with that aspect. In fairness to physicians, and, in fact, I invite their comments, I used to watch the literature in the journals that came in to our medical director. At the end of the week, there was always a stack that was at least a foot high. Now that clinician was required to be aware of everything published in all of that literature. I'm not sure that's humanly possible—in fact, I'm pretty sure it's not. It may be that it's up to people like us who specialize in an area to work as physician extenders to help these people to see these issues and to help them with their patients—particularly the general practitioner. I just can't imagine how any human being can possibly stay current with all aspects because, remember, we who are looking in this Journal Conference at only sleep-disordered breathing as one tiny aspect of sleep disorders, and we've identified all these areas of need that these general practitioners have the whole rest of the body to look at, with specialists in all the areas clamoring for them to have additional knowledge.

James: You were talking about being multidisciplinary and having various skills—that's actually been said several times in the conference. Keep in mind that not only are these people multidisciplinary, but also I don't think you have to worry too much about losing your best respiratory therapists. Remember, this is a horrendous job for the most part that involves a lot of things that you had listed, Pam, plus some other ones that I think weren't on your list, like patient counseling, hospitality, patient safety, physician liaison, and, of course, case manager, reimbursement specialist. All of this on a 12-hour overnight shift with no break and a salary that is probably not commensurate with what they're used to.

Minkley: It's important, too, to remember that just by the nature of "a sleep study" the physical location where this is done is often in, as they say, the 'bowels' of the hospital, or off-site. There's very little backup help unless you use a telephone to get someone out of bed. It's not like an acute care unit where you have physicians, residents, interns, nurses, and other respiratory care practitioners walking around. You're often sit-

ting by yourself or with one other person. Some of the challenges also include what I'm fond of saying is the "marriage aspect." We spend a lot of time dating and selecting our mate and choosing the person with whom we are to live, and, yet, when you look at the typical schedules in a sleep center you'll have a technologist shut in a small space for 12 hours at a time, 3 consecutive days, frequently, with another person whom they didn't date, they didn't select, but they have to live with them and operate closely in that environment. It brings a whole new meaning to 'leaving the cap off the toothpaste.' We have to deal with those social aspects of working together, too, in a very small, highly technical space.

Mathews: Can I get back to that question I deferred from yesterday? What are people teaching in the schools? We have 3 of us here from the schools. What's being taught? In terms of hours, let's say.

Barnes: We provide an extensive science background. If you were to review our curriculum, I think you'd be delighted with the number of basic and medical science courses. I believe it is the kind of background you would want for your sleep technologist. Our plans are to do a 2-week rotation in sleep labs to 'open the window' for this area of specialization to our students. We will encourage motivated students to seek out further postgraduate training.

Minkley: When you talk about basic sciences and physiology, when you talk about respiratory physiology, are there any presentations about respiratory physiology during sleep? Or are we only looking at the normal waking physiology?

Strollo: This is obviously a hot button here. The final comment should be from a respiratory care practitioner. So, Mr Mathews, you may close the conference.

Mathews: We may have the most sophisticated system for teaching sleep of the respiratory care programs. All of our students in their junior year get between 20-25 hours on sleep. 6-9 hours didactically and then 2 nights observational. In the senior year, we have what we call a senior specialty practicum, which is like a 1-semester internship. One of the options is sleep diagnostics. The students in this practicum get 16 more hours of didactics, and they spend 420-430 hours in a sleep lab. Our kids have been very successful in passing the RPSG exams and in getting jobs in sleep labs. I think that's unique because of the way we set up our program.

Minkley: I think we have a window of opportunity here that really shouldn't be closed. With the restructuring of the committees on respiratory care education, there is an opportunity to look at these aspects and incorporating these new ideas.

SEE PAGE 430

For a listing of sleep organizations in America

Sleep-Disordered Breathing: A Summary of the Conference

Patrick J Strollo Jr MD

History & Background
Hypopneas—Definition & Detection
Pathophysiology of SDB
Consequences of Sleep Apnea
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Challenges & Opportunities for Respiratory Therapists
In Summary

The impact of sleep-disordered breathing (SDB) on society and our health care system is substantial. That was highlighted by the presentations during the RESPIRATORY CARE Journal Conference on Sleep-Disordered Breathing and is reflected in the papers published here. As paradigms for health care delivery shift over the next few years, I believe that we will find that health care extenders, particularly respiratory therapists, can serve a useful role in providing high-level, 'high-tech' care for patients who have sleep-related breathing problems. In this Summary, I touch on the highlights of the Conference and focus some of my impressions, crystallizing some of the points that the speakers touched on.

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A version of this paper was presented by Dr Strollo at the close of the RESPIRATORY CARE Journal Conference on Sleep-Disordered Breathing, held in Cancún, Mexico, October 17-19, 1997.

History & Background

To launch the Conference, I provided some background on the history of the interest in SDB and sleep medicine, as did Pam Minkley. This is a field that is not vertically integrated in the manner that many core specialties are. Sleep medicine draws from psychiatry, from neurology, and more recently from pulmonology. Psychiatrists and neurologists first noticed breathing problems during sleep (as Minkley pointed out) and helped define sleep structure. When I first started in the sleep field, many, perhaps most, of the sleep community were from psychiatry and neurology. Now pulmonologists comprise the largest membership segment of the American Sleep Disorders Association. That shift has been driven primarily by the recognition of sleep apnea as a clinical problem, and the exponential growth in this field has been driven primarily by technology.

We sought to define SDB and particularly to make clear the distinctions between sleep hypopneas and the upper airway resistance syndrome. I want to make clear that I do not believe any current disagreement exists regarding sleep ap-

nea and its impact on mortality. Sleep apnea is easy to diagnose. The absence of air flow, whether detected by thermocouple, thermistor, nasal pressure transducer, or respiratory inductive plethysmograph, is easy to identify.

Hypopneas—Definition & Detection

Controversy revolves around sleep hypopnea without important nocturnal desaturation and the upper airway resistance syndrome. I believe that these entities are real and frequently merit treatment. A number of studies now suggest that patients experiencing them may benefit from treatment.[1,2] This has been my personal experience as well and is shared by many participating in the Conference. The problem is that we are left with the need to approach these patients with a trial of therapy. In a 1988 study published in *Chest,* He, Kryger, and colleagues[3] reported a much lower 8-year survival rate in OSA patients with apnea-hypopnea indexes > 20 than in those with indexes < 20. I believe that these data still hold up well. Kryger's study was retrospective, but a number of studies have corroborated it. When we blur the distinction between apnea and hypopnea, the impact on morbidity and mortality tends to be somewhat lessened. The other important 'take-home' point mentioned is that this effect may be more prominent in younger age groups.

We talked about the hypopnea detection threshold's being unclear in terms of impact on the scale of disease. I believe that we are in a mode now of 'fine tuning' our level of sensitivity for disease. This will be influenced by monitoring techniques that we use and the definitions that we formulate.

I believe that the future will bring a standard definition for hypopnea, fueled in part by the Sleep Heart-Health Study.[4] I think it will be useful for clinical purposes to have uniformity for respiratory monitoring techniques, much like standardization of spirometry by the American Thoracic Society. In the discussion session, Alex Chediak appropriately pointed out that one of the dangers of prematurely standardizing methodology is the possibility of impeding creativity. I think that we can continue to be creative, but we may need to do that in a little more rigorous fashion, with internal review board-approved protocols. We have had the luxury in sleep laboratories of performing research and incorporating new methodology into our clinical studies without having a standard. I think we have reached the point now at which standardization would be useful, so that the personnel in one laboratory can talk to those in another with some degree of assurance that they are speaking the same language and observing the same phenomena.

We also need to look for a better way of detecting arousal. Body movements with arousal are easy to detect, and they do correlate with daytime sleepiness. In fact, during the 5th International Sleep and Breathing Symposium, October 5-7, 1997, in Edinburgh, Scotland, the group from Oxford presented the results of an elegant study during which they looked at neural networking and pulse transit time and contrasted them with simple videotaping of body movement (unpublished data). The best correlation with daytime sleepiness was actual videotape and larger movements. So, the jury is out in terms of how to fine tune arousal detection, but I think we are well on our way.

Pathophysiology of SDB

The second talk of the Conference, the pathophysiology of SDB, was given by Alex Chediak. One of the things that always strikes me is, with the relatively small structures of the upper airway, how complex and intricate the interaction is. One of the reasons, as mentioned in the lecture, is that the upper airway in humans has multiple tasks: swallowing, singing, and vocalizing as well as breathing. As a result, a number of muscles—dilator and actuator muscles—are involved in these highly complex actions. SDB resulting in airway closure can be thought of as being mediated by a normal response to an abnormal airway. Many people with SDB have a small airway for one reason or another. A small group of patients has only compliance problems, but, in general, we are talking about a small airway. I think that is supported by data from Haponik.[5] When sleep apnea patients are contrasted with snorers and normal subjects, it can be seen that in those with sleep apnea the body is defending against a small airway to maintain airway patency, and that in both wakefulness and sleep this occurs because of activation of dilator muscles, particularly the best-studied genioglossus. Genioglossal muscle tone in those with sleep apnea is elevated compared to tone in normal subjects. Now, the problem that we get into is in addressing the concurrent influence of the sleeping state. When state-dependent changes are superimposed on compensatory response to airway opening, one finds that airway closure may ensue.

Chediak continued with a presentation of the current theory of the tube law and how airway closure is related to transmural pressure. Upstream and downstream resistance plus the components of tracheal tug and lung volume play a role in precipitating closure.

One of the interesting aspects of the progression of SDB is that people snore for a period of time and then, at some later date, develop sleep apnea. So, sleep apnea does not occur at the onset of snoring. That was described many years ago by Lugerisi[6] when he labeled this "heavy-snorers disease." A number of investigators studying this phenomenon find that it takes approximately 10 years of snoring before sleep apnea is detected. There are some 'holes' in some of the investigations, but that has been a general finding in a number of retrospective studies. One of the questions that arises is Does damage to upper airway receptors perpetuate and promote airway closure? One of the Swedish groups[7-9] has looked at the issue of oscillatory damage as a cause of neuropathy in the palate that leads to possible blunting of the compensatory response. So, we are beginning to understand that snoring starts and persists for a period of time, and, as people age the ongoing vibra-

tory trauma to the airway may actually establish the patient's propensity for airway closure.

Consequences of Sleep Apnea

The consequences of sleep apnea are, by no means, trivial. Kingman Strohl from Case Western outlined some of the main issues. Complications of SDB are mediated by sleep fragmentation and hypoxemia. The two cannot be investigated independently because, unfortunately, they are interrelated. Sleep fragmentation results in augmentation of sympathetic traffic and possible release of more and more epinephrine, which may be one of the primary determinants of high blood pressure in these individuals. This is undoubtedly affected by hypoxemia, and hypoxemia and hypoxic vasoconstriction may lead to pulmonary hypertension. The notion is that episodes of pulmonary hypertension over a period of time lead to fixed or diurnal hypertension. Of those patients found to have high blood pressure, not all normalize their blood when treated. The explanation for this has been that remodeling may occur, and if the causes remain untreated for a long period of time, hypertension persists.

Daytime sleepiness is one of the cardinal symptoms of SDB. It can be measured objectively as has been shown with multiple sleep latency testing, looking at the propensity to fall asleep, or with the maintenance of wakefulness test, examining the propensity to stay awake. Much of these data were first gathered by Milton Erman's group at the Scripps Clinic. A number of studies[10] have demonstrated that neural cognitive dysfunction occurs in these patients and is mediated by sleep fragmentation and hypoxemia. With regard to vascular abnormality most closely linked to SDB, many reports have identified stroke, myocardial infarction, and right ventricular failure to be associated with this diagnosis. As we discussed, these associations are based mostly on retrospective data, and, of course, methodologic problems emerge when one looks backwards. One of the things that we hope will define some of these risks is the sleep heart-health study, in which the plan is to follow patients prospectively through cohorts of approximately 6,000 patients. The degree of vascular risk incurred is not clear, and the question is not Does sleep apnea cause vascular risk? There seems to be no question about that in people with severe SDB. The issue that must be hammered out is What is the disease burden that results in vascular risk? When we are able to follow these cohorts with SDB, we should be better able to answer. We would like to establish the risk in order to provide patients with an idea of what the risk 'down the line' is if the condition is not treated. At the present time, if someone has an elevated apnea-hypopnea index but is asymptomatic and does not have high blood pressure, we believe that he or she is at risk for other vascular complications, but the data do not adequately support that right now. What we are left with is treating only symptoms that are disabling.

Who Should Be Treated Now?

There are patients who need to be treated now—patients with overwhelming subjective daytime sleepiness. They are unable to do the things that they need to do during the day, such as operating heavy equipment, driving an automobile, or being productive in the workplace. Objective daytime sleep testing with multiple sleep latency tests frequently identifies these individuals. Severe nocturnal desaturation mandates treatment and usually correlates with severity of the apnea-hypopnea index. We usually pick up these patients when their apnea-hypopnea indexes are > 40, and they are frequently symptomatic. Nocturnal angina occurs in a small subset of patients with SDB, and this subset probably comprises patients who have an additional burden in terms of vascular risk, such as somewhat trivial coronary artery disease. When a substantial hypoxic burden is imposed on someone with minor degrees of coronary artery disease, substantial ischemia in the ventricle may occur. When such patients are found to have sleep apnea, they benefit from therapy. Because of the high association between hypertension and SDB in patients who have poorly controlled high blood pressure,[11] one should think about the possibility of another mechanism's perpetuating the hypertension. If other risk factors suggest that sleep apnea is present, then one should possibly pursue nocturnal testing because control may be substantially improved if one treats the sleep apnea. One thing to remember is that there may be remodeling, and my experience and the experiences of others has been that all antihypertensive medications cannot be withdrawn even when the sleep apnea has been successfully treated.

Objective Measures of Sleepiness

The Epworth Sleepiness Scale[12] was developed as a research test, but I think it can be a helpful clinical tool to assess sleepiness objectively. The caveat is that a negative Epworth score may not be revealing, and the patient may be markedly sleepy. One of the things that we talked about during the Conference was that sleep apnea frequently presents in a subacute fashion. Patients make adjustments in their lifestyle and frequently fail to realize the severity of the problem—unlike gall bladder disease, for example, where a patient who has been well may be suddenly afflicted with a substantial problem. The point was made during Barbara Phillips's discussion of clinical diagnosis that fatigue does not equate to sleepiness. This is true and I agree. The problem is that patients frequently use the terms sleepiness and fatigue interchangeably. Sometimes, I find that teasing out fatigue and trying to have the patient define what is meant by fatigue can be helpful. Of interest, if you look at patients referred for chronic fatigue syndrome, one recent study indicated that 44% of patients studied with polysomnography were diagnosed with a sleep-related breathing disorder.[13]

Awareness & Identification

In terms of identification and in terms of clinical diagnosis, recognition is a major problem. This was recognized by the National Institutes of Health (NIH), and the publication of "Sleep Apnea: Is Your Patient at Risk?"[14] for primary care physicians and a parallel public information campaign are designed to increase awareness. One of the things that has grown out of the NIH initiative is the academic sleep award. Ed Haponik at Bowman Gray has devised an acronym I SNORED,[15] which he has demonstrated can be exported to primary care and medical student populations and appears to foster neural imprinting. The results of testing suggest that awareness can be increased and medical students do remember.

I — insomnia or insufficient sleep
S — snoring
N — not breathing
O — older or obese
R — restorative sleep
E — excessive daytime sleepiness
D — drugs and alcohol

Clinical Diagnosis & the Physical Examination

What is revealing in the physical examination? I do not think that we are yet speaking the same language in regard to the physical examination. A number of studies[16-18] suggest that the physical examination adds little, with the exception of weight and neck size as a function of upper body obesity. Many people describe the upper airway as being crowded or not crowded, but I think we can do a better job than that. The arch of the palate and the velopharynx may have an impact on nasal resistance. Looking at the soft palate and grading the palate according to the Mallampatti classification may help us in talking with our colleagues. Grading tonsils is important because 1+ tonsillar hypertrophy is different from 4+ hypertrophy in terms of the burden for airway closure—at least, in adults—although there may be some question in pediatric subjects. Intramaxillary and intramandibular distance affects the size of the 'box' and can impact on airway closure. I think we will see some interesting data emerge from the Stanford group, which has been looking at some simple ways to measure and better characterize the airway.

In patients with unexplained right heart failure or right heart failure out of proportion to the spirometric defect or right heart failure that does not improve with continuous oxygen supplementation in patients with chronic obstructive pulmonary disease, sleep apnea should be considered. It has been my personal experience that when you identify and treat sleep apnea in these patients, right heart function improves, and you can make an impact on their quality of life.

It should be clear that severe sleep apnea can be identified. There is no question about that, Those of us who spend a substantial amount of our time and our practice involved in caring for patients with sleep apnea are rarely surprised by severe sleep apnea. Patients who are symptomatic and have severe apnea can generally be easily identified. The conundrum right now relates to treating patients with milder disease. Will treatment truly benefit them?

Sleep Testing
In & Out of the Sleep Laboratory

Pam Minkley looked at some of the issues regarding testing in and out of the sleep center. Controversy currently exists with regard to the sensitivity and specificity of out-of-hospital or out-of-sleep-center testing. Some of the confusion probably has arisen from trying to apply this testing to groups that have not been preselected. In the future, I think we will be identifying subsets of patients who will be handled by different paradigms. Milton Erman pointed to that in his managed care scenario, the Sleep Physicians of California model. It is impossible for us, as sleep physicians, to take care of every single patient in the sleep laboratory. We must export some technology to the primary care setting to allow identification of patients who need to be treated. That will undoubtedly involve some form of abbreviated testing. Some of the testing will be diagnostic and will not require full polysomnography; split-night polysomnography may be reserved for patients who are symptomatic and desire immediate treatment.

People who have degrees of sleepiness that do not quite fit standard physical-examination criteria or in whom there is a question of other nonpulmonary sleep-related disorders may still best be handled in the laboratory. I think the future may involve more elegant analysis of EEG and breathing variables in those individuals. That will mandate adequate training for physicians and technicians. One of the things that I have been struck with over the past 10 years is that it takes more, rather than less, understanding to do abbreviated testing—to properly interpret one or two channels and know when you may not be getting an adequate assessment.

Positive Pressure Therapy

Charles Atwood from the University of Pittsburgh explained the theory and application of positive pressure therapy, the mainstay of treatment for sleep-related breathing disturbance. The reason that positive pressure is so effective, as it was pointed out by a number of participants, is because airway closure diffusely affects the unstable upper airway during sleep. If we provide a pneumatic split to the airway, we can stabilize the airway regardless of the site of closure.

Although continuous positive airway pressure (CPAP) is the standard time-tested therapy, we find that the procedure for titration varies from center to center. Some centers do not record pressure and flow patterns but rather titrate to adequate

oxyhemoglobin saturation (as reflected by pulse oximetry) and the abolition of EEG arousals. I believe that this may affect the nature or the accuracy of the titration. The American Sleep Disorders Association has not yet addressed a standard for CPAP titration. Charles Atwood addressed how we do things at the University of Pittsburgh, and I think we titrate in a fairly logical fashion. For our patients, titration is successful in 80%, a number that compares favorably with what we have found at other good centers throughout the country.

Bi-Level Pressure Application

I believe that considerable controversy exists regarding the role of bi-level pressure application. The use of bi-level pressure is based primarily on two papers in the literature—the original description of the BiPAP system by Mark Sanders of our group[19] and a second study that addressed the randomized application of bi-level pressure to a group of unselected patients with sleep apnea. In the second study, all comers with sleep apnea were treated with either CPAP or bi-level pressure. The finding was that in unselected patients compliance did not differ. There were some problems with the randomization scheme in that one study and a higher dropout rate in the CPAP arm than in the bi-level pressure arm. Where we have found bi-level pressure to be useful is as salvage therapy. Some abstracts but no papers have addressed this. Patients who have difficulty exhaling against a continuous pressure will not accept therapy if continuous pressure is used. Institution of bi-level pressure therapy may allow a subset of patients to be salvaged. I think there is a role for the bi-level treatment but clearly not in unselected patients but rather for salvage. The other group of patients who benefit from bi-level therapy are people who have concomitant hypoventilation. In those patients, a bi-level system provides ventilation as well as splinting of the airway.

Autotitrating Devices

The obvious 'new kid on the block' is the autotitrating device. As one reviews the literature, it is important to be aware that the computer algorithms in these devices are changing and improving. I have no doubt that autotitrating devices are going to occupy a place in our treatment armamentarium. Certain individuals experience little variation in airway closing pressure through the night. The need for an autotitrating device in those individuals is questionable. However, other individuals experience considerable variability, and in those people autotitration may improve compliance. This is a more complex technology than simple CPAP, and how robust it proves to be in use over time in responding to the patient remains to be shown. I think that is an engineering problem that can be worked out. Will use of autotitrating devices lower the cost of care? It may, if we are able to titrate outside of the laboratory and if follow-up studies are not required. The jury is out, but we hope this will be the case.

Patient Education & Partnering with Home Care

Karen Studnicki spoke on a topic that we at the University of Pittsburgh believe is extremely important—patient education. Patients need to be prepared for studies. They need to know what is going on. Patient education should be an ongoing process—as in any chronic illness. When we talk about providing a CPAP prescription on the basis of a full-night or a split-night study, we believe that involves not just providing a pressure prescription but also providing a proper interface. We have found that many patients fail to accept the therapy because of inadequate interfaces and substantial leaks. We change from continuous to bi-level pressure delivery if patients do not accept therapy. I believe, if one is to meet the needs of patients with sleep-related breathing disturbance, that is exceedingly important.

Once the proper prescription has been determined, the patient must accept therapy. If therapy is not accepted, one never reaches the point of gauging compliance. Studnicki shared some interesting data showing that compliance patterns may be determined within the first week of therapy. Perhaps we should target our ability to improve compliance in that first week! Now, on whether our interventions will be successful in irregular users (as opposed to regular users), I think the jury is out. It does appear that simple interventions that target patient education do favorably affect compliance as was reported by the Michigan group.

Nonsurgical Interventions

Larry Epstein pointed out that weight loss is an important nonsurgical intervention. Although weight loss is difficult to achieve, that does not mean that we should not counsel patients to try to lose. Even modest degrees of weight loss may lead to substantial improvement in airway function. Positioning is effective in the laboratory, but its long-term success rate is unclear. I find positioning useful in hospitalized patients. We try a combination of 30° elevation of the head and supplementation of oxygen while we are arranging to bring them to the laboratory for study. Data now support that 30° elevation is as effective as the lateral position and can be particularly useful in acutely ill hospitalized patients.

Medication can be a useful adjunct in the somewhat rare circumstance of the patient with concomitant hypothyroidism.

Oral appliances may be useful in mild disease, but they are expensive and are not 100% effective.

Surface electrical stimulation has not been shown to be very useful and may produce sleep fragmentation. The implantable electrodes that the group at Johns Hopkins has been working on for a number of years show some promise, but clearly the technology has not been perfected.

In the discussion following Epstein's talk, the question was asked Should residual sleepiness be treated with stimulants?

I think we will be seeing more about this. The medication modafinil has some interesting properties and less abuse potential and may fill a void for people who have been optimally treated but still complain of residual daytime sleepiness or have minimal degrees of desaturation. I think we will see some trials here in the U.S., studying this medication in some subsets of patients with SDB.

Surgical Interventions

Tucker Woodson described the variety of treatments involving upper airway reconstruction. I think we need to keep in mind that airway closure is a diffuse process, so that if one plans to operate on the upper airway, it is important to make sure that all areas of possible closure are addressed. Uvulopalatopharyngoplasty (UPPP) is a time-tested approach for the relief of snoring, and it does modify the degree of SDB. I do not believe there is much difference in outcome between the cold-knife technique and the laser other than that the laser procedure can be done in the outpatient setting. People think lasers are painless—a misconception. Any time you violate the mucosa, a substantial amount of pain results. Genioglossal advancement in people who have obstruction by the tongue has helped. Our experience has been similar to the experience of the Stanford group—coupling genioglossal advancement with UPPP is better than UPPP alone. Maxillary mandibular advancement—a 'big' surgery lasting 5 hours and requiring internal fixation—is highly effective in carefully selected patients. These surgeries tend to work much better in people who are closer to ideal body weight than in the obese, and Woodson shared with us the effect of body mass index on many of these.

Hyoid resuspension can improve airway patency and stabilize the airway. Tongue-base surgery in people who have large tongues can be useful. We will be hearing more about radio-frequency volume reduction. (We seem to be in volume-reduction mode in pulmonary medicine nowadays.) We are involved in a study looking at base-of-tongue volume reduction with this technique. The interesting thing about the radio-frequency technique (as contrasted with the laser technique) is that the mucosa is not violated, so there really is less pain. The problem, as Woodson pointed out, is with the cost of the devices—disposable devices costing $400-500 each, with treatment protocols requiring 4 to 5 treatments. That is a 'nice couch' that the patient has to pay for, but I predict that this treatment may assume a more prominent role.

SDB in Children

SDB in adults has been well funded with prospective SCOR grants, and we are now reaching the point at which funding needs to support looking at cohorts and following populations to help define SDB in children. Lee Brooks pointed out that diagnostic criteria for adults differ from criteria for children. In children, apneas may be shorter with marked desaturation because lung volumes are smaller. In some, because there is no concomitant lung disease, desaturation may not be present, but there may be increased work of breathing. These patients may present more with an upper-airway-resistance pathophysiology and may be affected negatively during the day. Some of these children may be misdiagnosed with attention deficit disorder. Tonsillar hypertrophy is what occurs to everyone, but tonsillectomy is not uniformly curative. My experience suggests that the sleep study is probably best done after the tonsillectomy, rather than before, to make sure that the patient is adequately treated, but, as Brooks pointed out, not everyone with tonsillar hypertrophy may need to be treated. CPAP is effective in children and children do well with this treatment.

The questions during Brook's discussion focused on Is adult sleep apnea a pediatric disease? Some data suggest that allergies in childhood may affect craniofacial growth, but one of the alternative explanations may be that craniofacial growth is most influenced by genetics—how the face is put together. The group at the University of California-San Francisco occluded the nares of monkeys at birth and noted that it did not appear to affect jaw development. This is clearly a subject that needs more funding so that we can better define the disease burden in this group, the diagnostic criteria, and possible lifelong ramifications.

Health Care Management & Sleep Medicine

Milton Erman pointed out that years of medicine practiced in a fee-for-service environment have resulted in mushrooming costs of health care and no economic responsibility of ownership by patients. I think he nicely summarized that. This has had a negative impact on our national debt and on the debt burden to many companies who foot the bill for health care. Managed care has promised increased efficiency and improved maintenance of quality and may change protocols for health care delivery and provide new standards of care. Those are the promises of managed care, but I do not believe that we have reaped those benefits yet.

Capitation is uncharted territory for the sleep medicine community. When one 'signs on' to deliver managed care, the increase in referrals may make survival difficult under capitation.

Erman also presented the novel idea that there may be a payoff on investment in our highly mobile society when patients move from one plan to another—not in terms of preserving health or for society in general but for the managed care organization.

Sleep medicine physicians have been able to do research within a variety of health care arenas, rather than just traditional university settings. We have been able to offset the cost of this research by additional revenue generated through patient care. This will become more difficult as health care dollars

become more scarce. We will need to rely on partnering with industry and on federal funding. Under such circumstances, we must make sure that quality keeps pace with quantity.

Challenges & Opportunities for Respiratory Therapists

Pam Minkley closed the presentations by voicing challenges and opportunities for therapists—one of the reasons for convening this Conference. The respiratory care provider is well positioned to be an integral part of the team that cares for patients with SDB. However, the respiratory care provider does need additional training. As participants have noted, some curricula address this, but many do not, failing to devote a substantial block to sleep apnea or SDB.

I believe that opportunities will evolve away from the hospital. In the future, the role of the hospital-based respiratory therapist will be focused primarily in critical and emergency care areas, with a consultant role likely for routine care. Respiratory therapists as knowledgeable providers may become involved in case management. There are opportunities in home care but also additional responsibilities. If therapists are to be involved in home care in sleep medicine, some integrated loop with the sleep center or the physician-director must be established as a mechanism to assure quality care for patients. There are many opportunities with the burgeoning technology. So I believe that respiratory care practitioners can participate. I believe that there are opportunities that will redefine job roles. Because of the 24-hour/day presence of therapists in the hospital, they can identify patients who desperately desire treatment.

In Summary

I believe that we can adequately identify patients with severe SDB—those with hypoxemia, high respiratory disturbance indexes, and marked daytime sleepiness. Current research is teasing out identification of those with milder disease and determining how they will benefit from treatment and what treatment is most appropriate. My Irish grandmother was want to say "If your only tool is a hammer, everything is a nail." If your only treatment is positive pressure, that may not be suitable for all patients with sleep-disordered breathing. I think that as we gain more sophistication if we are going to provide comprehensive services, we must include weight loss, smoking cessation, oral appliances, some upper airway reconstruction, and, possibly, surgical weight loss to best address the needs of these individuals.

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Dyspnea, No. 111, Lung Biology in Health and Disease, DA Mahler, Editor. Hardcover, 432 pages, illustrated. New York: Marcel Dekker Inc; 1998. $165.

This new volume, **Dyspnea,** in the series Lung Biology in Health and Disease takes a clinically-focused approach to the subject of dyspnea. The intended audience is physicians, respiratory physiologists, and therapists caring for clients with respiratory problems and those involved in pulmonary rehabilitation. In fact, the book's broad perspective on the subject makes it of interest to all health care professionals working with clients who are dyspneic.

The book's content reflects the beginning shift in medicine away from an emphasis on physiologic causes of the symptom and potential technologic or pharmacologic solutions toward client-centered assessment and outcomes. It opens with two chapters—Chapter 1—Dyspnea: Sensation, Symptom, and Illness and Chapter 2—The Language of Dyspnea—focusing on the symptom of dyspnea in the context of the total illness experience and as a symptom with a distinctive language for the different conditions in which it occurs. The physiologic basis of dyspnea is reviewed in detail in Chapters 3—Mechanisms of Dyspnea and 4—Exertional Breathlessness in Chronic Respiratory Disease. Chapters 5—Clinical Measurement of Dyspnea and 6—Dyspnea and Quality of Life in Chronic Obstructive Pulmonary Disease address the thorny issues of how to measure symptom severity in the clinical setting and during exercise, and the role of dyspnea in influencing clients' perceptions of their health status.

In the most traditionally medically-oriented section of the book, diagnosis is discussed in Chapter 7—Diagnosis of Dyspnea. Pharmacologic and technologic treatments are presented in Chapters 8—Strategies in the Treatment of Dyspnea: Disease Specific and 10—Treatment of Dyspnea: Physical Modalities, Oxygen, and Pharmacology. In contrast, Chapter 9—Coping Strategies for Dyspnea builds more directly on the broader view of dyspnea presented in the first two chapters in that it presents cognitive-behavioral interventions aimed at the symptom in the total illness context and as it is perceived by the individual experiencing it. In a final chapter that underscores the comprehensive coverage of the symptom in this book, evaluation and treatment of dyspnea in clients on mechanical ventilation are examined in Chapter 11—Dyspnea in the Ventilator-Assisted Patient.

In many ways, this volume represents an update of Mahler's earlier volume on dyspnea.[1] As such, it draws on the most recent research and provides extensive references for the interested reader to pursue. The book is generally well written and well organized, although there are instances within individual chapters that are exceptions. In particular, the chapter on clinical measurement of dyspnea is organized in a counter-intuitive way. It makes sense to present multiple instruments in each of two categories: clinical scales and ratings during exercise. However, the presentation of psychometric data (reliability, validity, responsiveness) is difficult to follow, because the groups of instruments are discussed together ie, reliability of all of the clinical instruments is discussed in one section, followed by validity of all the instruments, and finally, responsiveness of all of them). The chapter would have read more clearly had individual instruments within types been discussed separately or had the psychometric data been presented in tabular form.

To the editor's credit, the usual limitations of edited books, in particular redundancy and overlap between chapters, have been minimized in this book. Most of the limitations of this volume reflect the fact that knowledge is incomplete or data are inconclusive in many areas related to dyspnea. Although much is known about the physiology of breathing, the exact mechanisms for producing dyspnea in different diseases or under varied conditions are unknown. Many of the innovative therapies proposed, whether technologic-pharmacologic or cognitive-behavioral, are not yet supported by empirical data. The definitive volume on dyspnea awaits further developments in the physical and human sciences that will allow us to treat dyspnea as successfully as we have treated pain. In the meantime, this book provides clinicians with an excellent resource to stimulate new ways of thinking about dyspnea and to guide treatment of the symptom as part of a larger illness experience.

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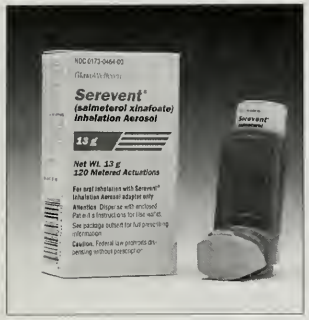

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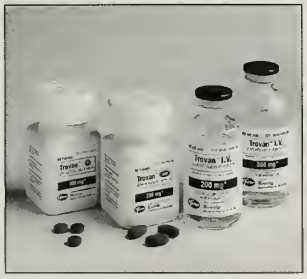

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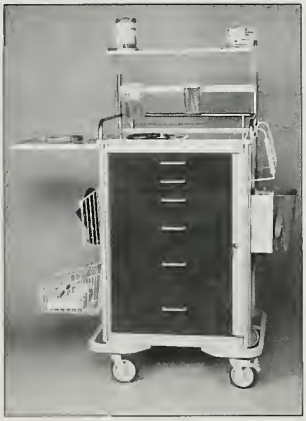

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RRT Examination	June 6, 1998 Application Deadline: February 1, 1998	120 Written only (new applicant) 80 Written only (reapplicant) 130 CSE only (all applicants)
	December 5, 1998 Application Deadline: August 1, 1998	250 Both (new applicant) 210 Both (reapplicant)
CPFT Examination	June 6, 1998 Application Deadline: April 1, 1998	130 (new applicant) 100 (reapplicant)
RPFT Examination	December 5, 1998 Application Deadline. September 1, 1997	180 (new applicant) 150 (reapplicant)
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___ lbs
or
___ kgs

B. Adverse event or product problem

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☐ required intervention to prevent permanent impairment/damage
☐ other.

6. Date of event (mo/day/yr)

7. Date of this report (mo/day/yr)

8. Describe event or problem

9. Relevant tests/laboratory data, including dates

10. Other relevant history, including preexisting medical conditions (e.g., allergies, race, pregnancy, smoking and alcohol use, hepatic/renal dysfunction, etc.)

C. Suspect medication(s)

1. Name (give labeled strength & mfr/labeler, if known)
#1
#2

2. Dose, frequency & route used
#1
#2

3. Therapy dates (if unknown, give duration)
from/to (or best estimate)
#1
#2

4. Diagnosis for use (indication)
#1
#2

5. Event abated after use stopped or dose reduced
#1 ☐ yes ☐ no ☐ doesn't apply
#2 ☐ yes ☐ no ☐ doesn't apply

6. Lot # (if known)
#1
#2

7. Exp. date (if known)
#1
#2

8. Event reappeared after reintroduction
#1 ☐ yes ☐ no ☐ doesn't apply
#2 ☐ yes ☐ no ☐ doesn't apply

9. NDC # (for product problems only)
– –

10. Concomitant medical products and therapy dates (exclude treatment of event)

D. Suspect medical device

1. Brand name

2. Type of device

3. Manufacturer name & address

4. Operator of device
☐ health professional
☐ lay user/patient
☐ other.

5. Expiration date (mo/day/yr)

6.
model #
catalog #
serial #
lot #
other #

7. If implanted, give date (mo/day/yr)

8. If explanted, give date (mo/day/yr)

9. Device available for evaluation? (Do not send to FDA)
☐ yes ☐ no ☐ returned to manufacturer on ___ (mo/day/yr)

10. Concomitant medical products and therapy dates (exclude treatment of event)

E. Reporter (see confidentiality section on back)

1. Name & address

phone #

2. Health professional?
☐ yes ☐ no

3. Occupation

4. Also reported to
☐ manufacturer
☐ user facility
☐ distributor

5. If you do NOT want your identity disclosed to the manufacturer, place an " X " in this box. ☐

FDA

Mail to: MEDWATCH
5600 Fishers Lane
Rockville, MD 20852-9787

or FAX to:
1-800-FDA-0178

FDA Form 3500 1/96)

Submission of a report does not constitute an admission that medical personnel or the product caused or contributed to the event.

ADVICE ABOUT VOLUNTARY REPORTING

Report experiences with:

- medications (drugs or biologics)
- medical devices (including in-vitro diagnostics)
- special nutritional products (dietary supplements, medical foods, infant formulas)
- other products regulated by FDA

Report SERIOUS adverse events. An event is serious when the patient outcome is:

- death
- life-threatening (real risk of dying)
- hospitalization (initial or prolonged)
- disability (significant, persistent or permanent)
- congenital anomaly
- required intervention to prevent permanent impairment or damage

Report even if:

- you're not certain the product caused the event
- you don't have all the details

Report product problems – quality, performance or safety concerns such as:

- suspected contamination
- questionable stability
- defective components
- poor packaging or labeling
- therapeutic failures

How to report:

- just fill in the sections that apply to your report
- use section C for all products except medical devices
- attach additional blank pages if needed
- use a separate form for each patient
- report either to FDA or the manufacturer (or both)

Important numbers:

- 1-800-FDA-0178 to FAX report
- 1-800-FDA-7737 to report by modem
- 1-800-FDA-1088 to report by phone or for more information
- 1-800-822-7967 for a VAERS form for vaccines

If your report involves a serious adverse event with a device and it occurred in a facility outside a doctor's office, that facility may be legally required to report to FDA and/or the manufacturer. Please notify the person in that facility who would handle such reporting.

Confidentiality: The patient's identity is held in strict confidence by FDA and protected to the fullest extent of the law. The reporter's identity, including the identity of a self-reporter, may be shared with the manufacturer unless requested otherwise. However, FDA will not disclose the reporter's identity in response to a request from the public, pursuant to the Freedom of Information Act.

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200 Independence Avenue, S.W.
Washington, DC 20201

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FDA Form 3500-back

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Drug Capsule: A mini-review paper about a drug or class of drugs that includes discussions of pharmacology, pharmacokinetics, and pharmacotherapy.

Graphics Corner: A brief case report incorporating waveforms for monitoring or diagnosis—with Questions, Answers, and Discussion.

Kittredge's Corner: A brief description of the operation of respiratory care equipment—with information from manufacturers and editorial comments and suggestions.

PFT Corner: Like Blood Gas Corner, but involving pulmonary function tests.

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Test Your Radiologic Skill: Like Blood Gas Corner, but involving pulmonary medicine radiography and including one or more radiographs; may involve imaging techniques other than conventional chest radiography.

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Article in a publication that numbers each issue beginning with Page 1:

> Bunch D. Establishing a national database for home care. AARC Times 1991;15(Mar):61,62,64.

Corporate author journal article:

> American Association for Respiratory Care. Criteria for establishing units for chronic ventilator-dependent patients in hospitals. Respir Care 1988;33(11):1044-1046.

Article in journal supplement: (Journals differ in their methods of numbering and identifying supplements. Supply sufficient information to promote retrieval.)

> Reynolds HY. Idiopathic interstitial pulmonary fibrosis. Chest 1986; 89(3 Suppl):139S-143S.

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> Enright P. Can we relax during spirometry? (editorial). Am Rev Respir Dis 1993;148(2):274.

Editorial with no author given:

> Negative-pressure ventilation for chronic obstructive pulmonary disease (editorial). Lancet 1992;340(8833):1440-1441.

Letter in journal:

> Aelony Y. Ethnic norms for pulmonary function tests (letter). Chest 1991;99(4):1051.

Paper accepted but not yet published:

> Hess D. New therapies for asthma. Respir Care (year, in press).

Personal author book: (For any book, specific pages should be cited whenever possible.)

> DeRemee RA. Clinical profiles of diffuse interstitial pulmonary disease. New York: Futura; 1990. p. 76-85.

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> American Medical Association Department of Drugs. AMA drug evaluations, 3rd ed. Littleton CO: Publishing Sciences Group; 1977.

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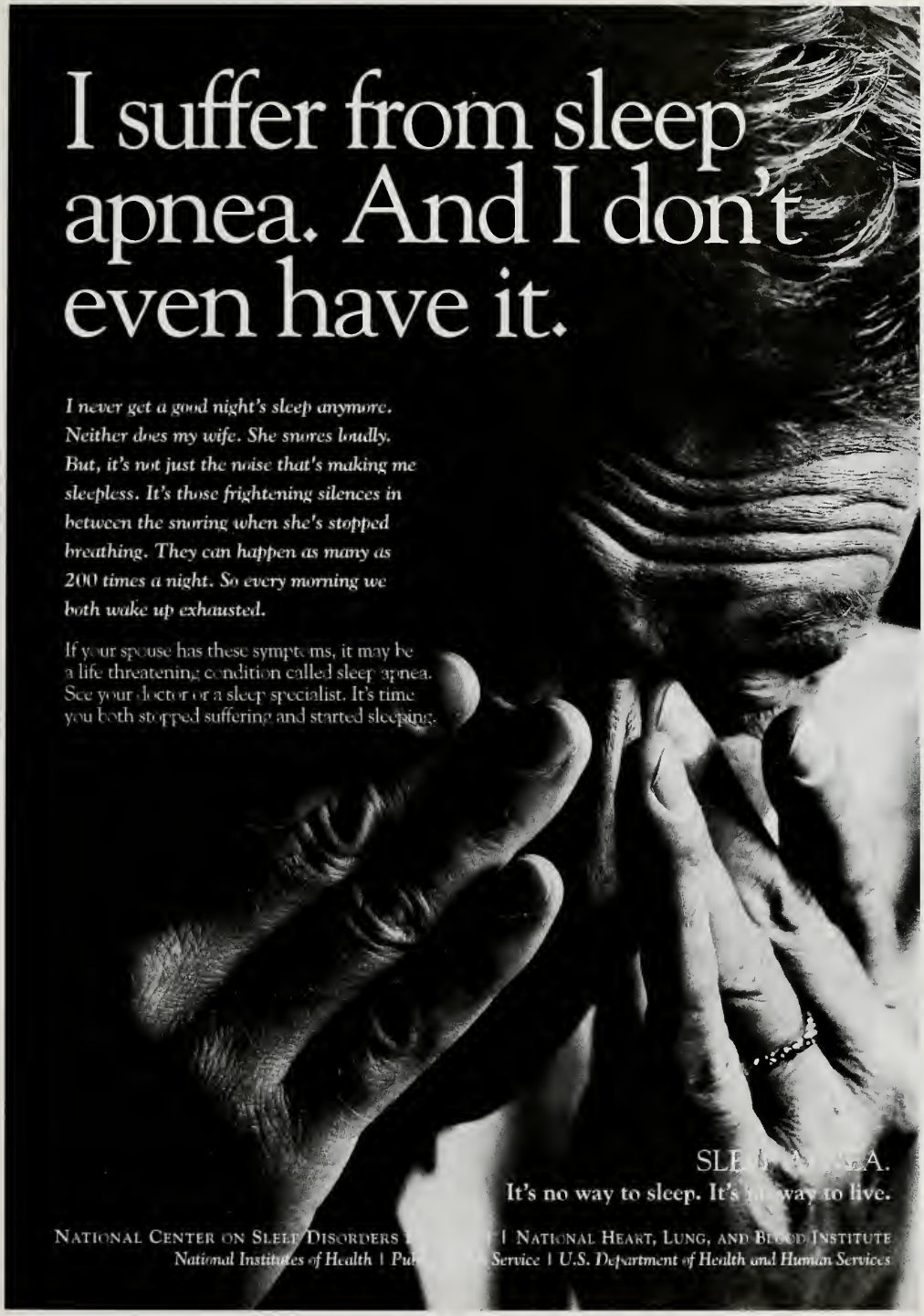
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View *Pulmonary Rehabilitation*, Part 3 of the AARC's 1998 Professor's Rounds Program Series, & participate in a live telephone question-and-answer session.
CRCE: 1 credit hour.
Contact: The AARC—(972) 243-2272 to receive the 90-minute video & register.

May 12–15 — *Region II of the Indiana, Kentucky, and Ohio Societies*
25th Annual Region II Conference, Hyatt Regency Hotel & Albert B Sabin Convention Center, Cincinnati, Ohio.
Contact: (800) 691-3046, Mail Box #3; web: **www. bright.net/~dsibb/reg2rc.htm**

May 20–22 — *Southwest Region of the Texas Society*
27th Annual Respiratory Care Symposium & Technical Exhibition, El Paso Community College, Transmountain Campus, El Paso, Texas.
CRCE: 12 to 16 hours are available.
Contact: Yvonne Banegas—(915) 577-6563.

May 22 — *University of Texas Health Science Center at San Antonio, Texas Society (Alamo District), & Wilford Hall Medical Center*
3rd Annual Riverwalk Respiratory Care Symposium, San Antonio Marriott Riverwalk Hotel, San Antonio, Texas.
CRCE: 6 credits available.
Contact: (210) 567-3706.

June 12–14 — *AARC Patient Assessment Course*
Atlanta Airport Marriott Hotel, Atlanta, Georgia. Preregistration is required; See page 362-363 for an application.
CRCE: 16 credits available.
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June 16 — *Videotape Teleconference*
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June 25–27 — *California Society*
30th Annual Convention—Thirty Something: Three Decades of Progress & Change—Monterey Marriott Hotel, Monterey, California.
CRCE: 17 hours available.
Contact: (805) 723-1180.

September 16–18 — *Maryland/ District of Columbia Society*
18th Annual Conference by the Sea, Sheraton in Ocean City, Ocean City, Maryland. Two certification programs—*Advanced Respiratory Skills for Skilled & Subacute Care* and *Patient Assessment*—will also be offered.
Contact: Jeanette Ledbetter—(202) 574-6348.

OTHER MEETINGS

May 15–16 — *American Society for Testing Materials & EMS Magazine Exposition*
ASTM Committee F-30 on Emergency Medical Services, Nashville Convention Center, Nashville, Tennessee.
Contact: Len Morrissey—(610) 832-9730; e-mail: lmorriss@ astm.org.

May 21 — *Respiratory Care Services of Arkansas Children's Hospital*
Diamond Conference—Annual Pediatric/Neonatal Cardiopulmonary Critical Care Conference, Little Rock, Arkansas. Forrest M Bird MD will be the keynote speaker; also a special session for nurses.
CRCE: Category 1 credits, contact hours for nurses, & physician CMEs are available.
Contact: Rachel Bull—(501) 320-3530; fax (501) 320-1887;e-mail: manders@exchange.ach.uams.edu; web; **www.ach. uams.edu/announce/diamond/**

July 9–11 — *Medical Graphics Corporation*
Cardiopulmonary Diagnostics 1998: Pathways to the Future, San Antonio, Texas.
Contact: (800) 950-5597, ext 199.

October 8–10 — *Medical Graphics Corporation*
Cardiopulmonary Diagnostics 1998: Pathways to the Future, Anaheim, California.
Contact: (800) 950-5597, ext 199.

August 7–9 — *American Lung Association of Southeast Florida, American Heart Association, & Florida Association of Cardiovascular & Pulmonary Rehabilitation*
4thannual Cardiopulmonary Rehabilitation Conference, Jupiter Beach Resort, Jupiter, Florida.
CRCE: Credits are available.
Contact: Carol Boyd—(561) 659-7644 by July 31

September 18–20 — *ELSO*
1998 Annual ELSO Meeting, Placido del Rio Hilton, San Antonio, Texas.
Contact: Phoebe Hankins—(734) 998-6600; fax: (734) 998-6602; e-mail: phankins@umich.edu; web: **www.med.umich. edu/elso**

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July 17–19, 1998
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